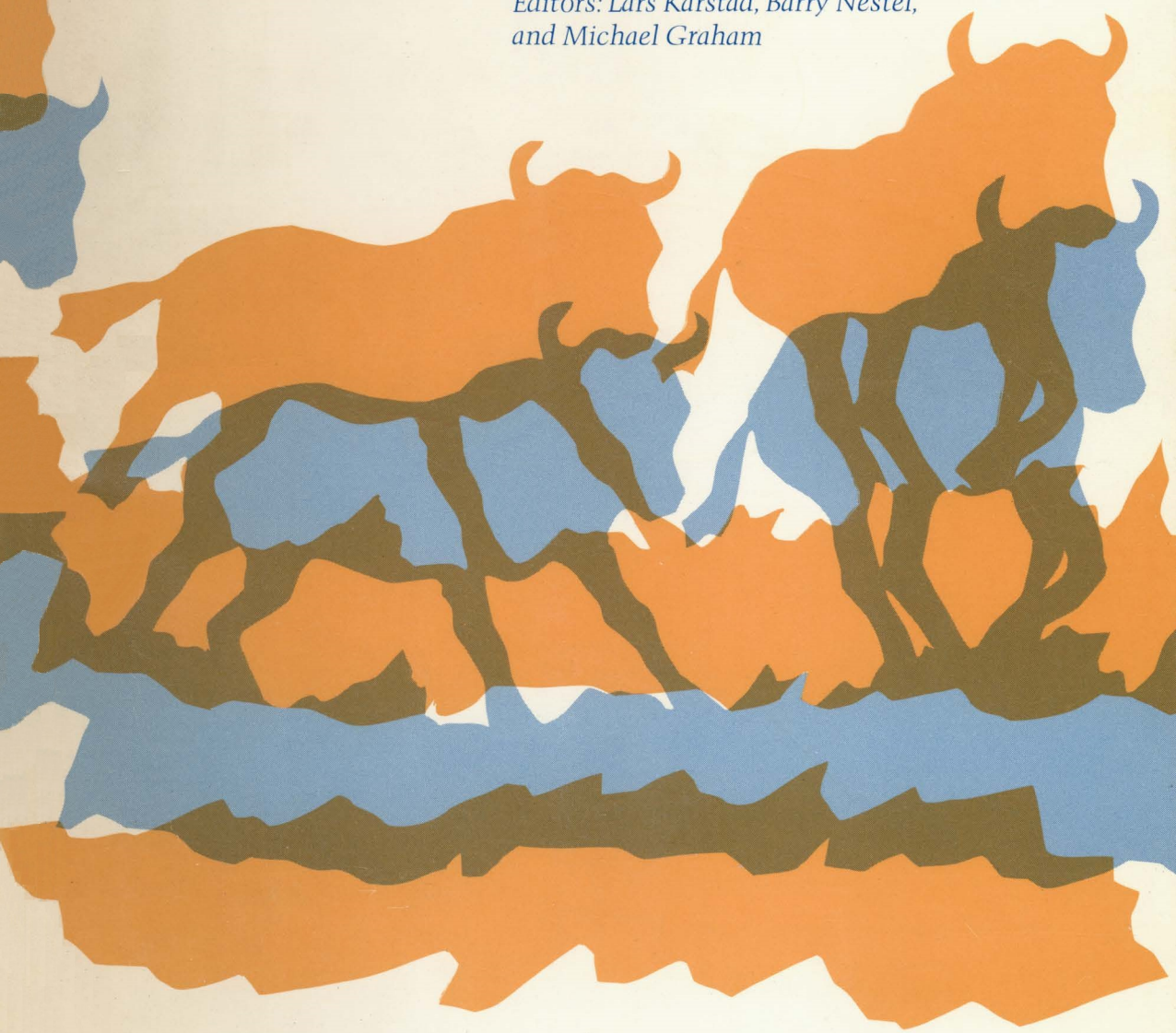
Wildlife Disease Research and Economic Development

Proceedings of a workshop held in Kabete, Kenya, 8 and 9 September 1980

Editors: Lars Karstad, Barry Nestel, and Michael Graham



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Contents

Foreword

For many years wildlife have been thought to be carriers of the causative agents of a number of diseases of domestic livestock in Africa. Efforts to protect domestic ruminant populations have led to the slaughter of many thousands of wild ruminants, even though their role in the epidemiology of diseases of domestic ruminants was often not clearly established.

During recent years, a number of changes have taken place in the pattern of land use in East Africa that have made it important to gain a better understanding of the disease relationships between domesticated and wild stock. One of the most important factors contributing to this need is the significance that wildlife now have in the economies of certain countries because of the tourist revenue that is earned through game parks.

In several countries, changes in the pattern of land tenure have been associated with the development of group or communal ranching. This pattern of changes seeks to improve the utilization of grazing and water resources and sometimes leads to competition between domesticated and wild animals. Thus, appropriate knowledge for maximizing the complementarity and minimizing the competition between the two groups of animals is important.

During the late 1960s, FAO (Food and Agriculture Organization of the United Nations) established a project with the Government of Kenya to carry out research on wildlife diseases. This project ran for 8 years and was then superseded by a similar type of project supported by the Government of Canada through the Canadian International Development Agency (CIDA).

The latter project had three specific objectives: to survey naturally occurring wildlife populations for the causative agents of certain specific diseases; to test the pathogenicity and transmissibility of these diseases in experimental animals both domestic and wild; and to train African veterinarians and support staff in epizoological techniques and basic wildlife disease research.

There was considerable continuity of staffing between the FAO and the CIDA projects and the Wildlife Diseases Section of the Veterinary Research Laboratories at Kabete now has 13 years of experience in carrying out research on wildlife diseases. That experience has been considerably enhanced by a widespread program of collaboration with a large number of other agencies involved in veterinary and zoonotic disease research in Kenya.

Since the research has now been under way for several years, the International Development Research Centre (IDRC), who manages the CIDA support for the research, agreed with the Veterinary Department of the Government of Kenya to sponsor a 2-day workshop to examine the implications of the research in terms of economic development.

The workshop was held in the conference facilities of the International Laboratory for Research on Animal Diseases (ILRAD) at Kabete on 8 and 9 September 1980. The first day of the meeting consisted of a series of short presentations that dealt with specific diseases. These presentations covered foot-and-mouth disease, rinderpest, malignant catarrhal fever, arboviruses, rabies, bovine petechial fever, theileriosis, trypanosomiasis, nematodiasis, echinococcosis, and cysticercosis. Each of the authors spoke of the relationship between domestic and wild animals. The implications of the specific diseases with respect to the development of both the domestic and the wild animal industries were also covered.

On the second day of the workshop the overall value of the research findings was considered from the point of view of the veterinary research department, the livestock producer, the wildlife rancher, and the ecologist. A general discussion on these papers helped to present an overall perspective of the economic role of wildlife disease research.

The workshop was timed to follow a large international gathering at ILRAD and a number of participants stayed over so that, although the discussions had primarily a Kenyan focus, the participants in the workshop included representatives of a number of West and East African countries, Europe, and North America. A particularly encouraging feature of the meeting was the participation of so many different institutions and individuals working in Kenya whose common interests overlapped with the theme of the workshop.

It is hoped that this report will serve to focus attention on the complementary role that domesticated and wild animals can play in terms of land utilization and economic development and that this will lead to a continuing interest in wildlife disease research now that its economic significance has been so clearly demonstrated.

IDRC is indebted to the Director General of ILRAD for providing conference and dining facilities to the workshop participants. Particular thanks are also due to Drs Chema, Brocklesby, and Ngulo for chairing the meeting sessions and to Drs Woodford, Zwart, Taiti, and Huhn for their leadership in the discussion sessions. Thanks are also due to Drs Karstad and Grootenhuis who undertook the responsibility for most of the planning and groundwork that ensured a successful meeting.

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Opening Address

S. Chema[1]

It is my pleasure, on behalf of my ministry, to welcome you to this workshop. The aim is not only to present and discuss scientific papers but to highlight the wider implications of research findings.

The central theme of our current development plan is the alleviation of poverty through the creation of employment opportunities and the development of the arid and semi-arid areas of Kenya. Development in the small-holder sector is progressing rather well, but much remains to be done in the rangelands.

In an effort to develop the drier regions of Kenya the government has embarked on a number of research and development programs geared specifically to these areas. These include the Livestock Development Project, the Dryland Farming Research Project, the Range Research Expansion Project, various Integrated Development Projects, and other programs for the arid and semi-arid zones. The Wildlife Diseases Research Project should be viewed as a project aimed at developing the rangelands that, in Kenya, support wildlife as well as livestock.

The idea of African wild animals harbouring parasites and diseases transmissible to man and his domestic animals is not new. In a book published in 1857, David Livingston said of the tsetse fly: "The bite of this poisonous insect is certain death to ox, horse and dog... [but] harmless to man and wild animals." He suggested that destruction of game would eliminate the flies by removing their food supply. The ineffectiveness of this apparently simple solution was amply demonstrated, but not before the needless slaughter of thousands of wild animals. Attempts to control rinderpest also resulted in the deaths of many thousands of wild animals. For example, almost 10 000 animals were killed between 1941 and 1951 along a 167 mile (270 km) section of the Tanganyika border with Northern Rhodesia (Vaughn-Jones 1953). We were told in Kenya that efforts to control rinderpest by vaccination would be meaningless unless it was possible to vaccinate the wild ungulates that abound in this country. Fortunately, time has proven that it is unnecessary and unjustified to institute wholesale slaughter of game to control tsetse or rinderpest. It is these activities, however, that have made some wildlife conservationists wary and suspicious of those who study wildlife diseases.

This meeting may help to dispel some of that distrust. Although some investigations were started in Kenya much earlier, it was not until the early 1960s that much emphasis was placed on the need for research into the role of wild animals as reservoirs and transmitters of diseases of importance to livestock production and human health. In 1961, a "Conference on Land Management Problems in Areas Containing Game" was held at Lake Manyara, Tanganyika. Papers on diseases, most of them presented by workers at the East African Veterinary Research Organization, Muguga, and by workers from the Veterinary Research Laboratories, Kabete, figured prominently on the program. There were papers on rinderpest by Scott and Plowright; on malignant catarrhal fever by Plowright; on African swine fever by DeTray; on theileriosis by Brocklesby; on ticks by Walker; on tsetse by Langridge; and on other parasites by Barnett. The proceedings were summarized in the East African Agriculture and Forestry Journal by Pereira (1961). A project, specifically for the study of diseases of wild animals, was established at the Veterinary Research Laboratories, Kabete, in 1967. At first supported by the Food and Agriculture Organization of the United Nations, this project is currently funded by the Canadian International Development Agency (CIDA). It is the CIDA-supported project that has grown into the Wildlife Diseases Section, which is presently staffed by three Kenyan Veterinary Research Officers and two expatriate wildlife disease specialists. This workshop provides evidence of the fruitful collaboration that occurs between our staff and the staff of several other institutions, including our host, the International Laboratory for Research on Animal

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Diseases (ILRAD). We are grateful to ILRAD for making these facilities available.

The research topics that we will be discussing in the next day and a half have been selected mainly for their relevance to livestock production. In the course of these studies, many other tempting research subjects have been seen. Some of these have been explored to a limited extent to determine their relevance to development goals. The point I want to make is that the list of diseases covered in this meeting is certainly not exhaustive; many other diseases and parasites demand research.

The recent reorganization of our ministry has emphasized our increasing concern with all aspects of livestock production, not just diseases and parasites. We must be increasingly concerned with animal nutrition, animal reproduction, range management, and land-use planning. Wild animals can also be regarded as "livestock" by the farmer and rancher. In some arid and marginal lands poorly suited to traditional livestock production, it may be possible to manage certain species of wild animals to obtain an economic return not only from viewing but also from using the animals as a protein source. As well, in tick- and tsetse-ridden areas, the production of wild animals, with their greater resistence to tick- and tsetse-borne diseases, may produce greater economic returns.

Our concern has been, first, the influence of wild animals and their parasites upon domestic livestock, and second, their influence on human health. But, what about the effects of disease upon the wild animals themselves? This question is largely unanswered. It is our hope that in the future our facilities and manpower resources will be such that we can study the effects of parasites and diseases upon the wild animals themselves.

A modern trend in veterinary medicine is herd health — a concern for the ways and means of improving the health of herds and populations of animals rather than of the individual animal. This population medicine approach can be applied to wild animals. The increasing pressure on land for various human uses, means that its use by wild animals will become restricted and that in parks, reserves, and game ranches, the use of land by wild animals will be intensified. When we restrict the movements of living beings and crowd them together, whether they be people, pigs, or chickens, new disease problems appear. The same is sure to happen with wild animals. We can no longer say "Let Nature take its course" because we have so changed the environment and so modified the freedom of the wild animals that Mother Nature is no longer with us! Diseases rarely have only one cause. Nutritional deficiencies may allow parasites to have greater effects, and problems of over-population, over-use of habitat, or soil contamination by the constant presence of animals, allow parasites to build up in number.

We are concerned about the health of Kenya's wildlife and we realize that wild animals are among our greatest natural resources. The presence of a number of biologists and wildlife managers indicates that we have many interests in common. We look forward to even greater cooperation in future.

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The Role of Wildlife Disease Research in Livestock Development

Lars Karstad[1] and Barry Nestel[2]

This workshop is somewhat unusual in that we are going to talk about fairly sophisticated technology in the context of its developmental role. Thus, we will focus not on blood counts or serological titres but on the relationship of certain major epizootic and zoonotic diseases to livestock development and human welfare and to the natural resources of the country. Perhaps we could clarify this a little by explaining how the Canadian Government became interested in supporting wildlife disease research.

The Pioneer Ranch Development Scheme in the Masai area was begun by the World Bank and the Swedish Government (SIDA) in the late 1960s. The scheme was designed to establish rational utilization of rangelands by controlling grazing and by improving disease control and access to water. The scheme was complex because it had to take into consideration the interrelationship between domestic stock and the abundant natural fauna as any substantial increase in domestic animal numbers would automatically lead to an encroachment on land used predominantly by game.

The preservation of the wildlife population is important because it is a considerable tourist attraction and a source of a large part of Kenya's foreign exchange earnings. Any major disturbance in the balance of the relationship between domestic and wild animals could endanger the growth of the livestock and/or the tourist industries.

The experiences gained in the Masai ranching scheme and the need to develop Kenya's large and latent livestock industry encouraged a consortium of donor agencies to offer the Kenya Government support for a second stage of ranch development in the mid 1970s. This consortium comprised the World Bank and a number of national development agencies, including the Canadian International Development Agency (CIDA).

Part of the CIDA funding was allocated to the Kenya Rangeland Ecological Monitoring Unit (KREMU), which was designed to provide information on the degree of competition for grazing and water between domesticated and wild animals. This knowledge would facilitate the establishment of sound strategies for formulating appropriate stocking rates on new ranches and for determining the need for, and location of, new watering points. In addition, the Canadian Government responded to a request from Kenya for support for strengthening wildlife disease research. This request was based upon the common susceptibility of domestic stock and wildlife to certain diseases. Wildlife can act as hosts to some bacterial, viral, and protozoal diseases that can be transmitted to domestic livestock. The transmission of these diseases is, in many cases, enhanced by the large ectoparasite population found in Africa. In some areas, cattle may be absent because of the presence of diseases which are harboured by wildlife and which may cause high mortality in exposed cattle. However, the literature on this subject is often confusing and controversial and much of the work was done some years ago before many of the current techniques in immunology and serology had been properly developed. In view of the importance of both cattle and wildlife to the economy of Kenya, it is important to verify as many of the uncertainties as possible in order to optimize the development of both industries.

The Wildlife Diseases Section of the Veterinary Research Laboratories at Kabete, Kenya, has made considerable progress toward such clarification. The section has been particularly fortunate in that it has not had to work in isolation but has been able to collaborate with a large number of national and international agencies working in the veterinary and wildlife fields in Kenya. Our partners in this work include members of the staff of the Veterinary Research Laboratories; the International Laboratory for Research on Animal Diseases (ILRAD); the Veterinary Department of the Kenya Agricultural Research Institute (KARI); the Food and Agriculture

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Organization of the United Nations; the World Health Organization; the University of Nairobi; the University of Guelph; the University of Utrecht; the University of Leiden; the University of Alberta; the Commonwealth Institute of Helminthology; and the many farmers, ranchers, and landowners in Kenya on whose land we have worked, notably David Hopcraft, Gilfred Powys, and the Galana Game and Ranching Company.

The research work of the Wildlife Diseases Section and its collaborators has concentrated on two specific objectives: to survey naturally occurring wildlife populations for the causative agents of certain specific diseases and to test the pathogenicity and transmissibility of these diseases in experimental animals (both domestic and wild).

As examples, several of the approximately 30 wild ruminant species found in Kenya have been examined for trypanosomiasis and of these the giraffe and the buffalo have been shown to be potentially important carriers (Karstad et al. 1978). Buffalo are the only wild animals found so far to carry *Theileria* pathogenic to cattle but *Theileria* species have been found in other wild ruminants although their significance is not fully understood (Grootenhuis 1979). The wildebeest has long been known to carry malignant catarrhal fever (Plowright et al. 1960) and bushbuck to be associated with bovine petechial fever (Snodgrass et al. 1975). The buffalo is also the only wild ruminant commonly infected with foot-and-mouth disease although virus from carrier buffalo is not readily transmitted to cattle (Anderson et al. 1979).

As an example of a disease believed to have been imported into Africa with domestic livestock, rinderpest took a very heavy toll of native wild ruminants in the early 1900s. Contrary to expectations, it does not seem to have become established in the wildlife, at least not as the virulent disease seen earlier (Scott 1970).

A study of intestinal parasites of sheep showed that although Thomson's gazelle could carry certain species transmissible to sheep, the gazelle did not appear to be an important source of infection for sheep. This is significant because sheep and gazelle often graze together (Preston et al. 1979).

At risk of oversimplification, we can divide the diseases of wildlife and domestic animals into four broad categories:

(1) Diseases of domestic animals not known to be naturally transmissible to wildlife. Examples of such diseases are the contagious bovine and caprine pleuropneumonias, bovine babesiosis (red water), and Nairobi sheep disease. In this workshop we do not need to concern ourselves further with diseases in this category.

(2) Diseases of domestic animals, initially exotic to Africa, that have invaded the indigenous fauna. Rinderpest is a prime example.

(3) "African diseases," in other words, infections and parasites occurring primarily in indigenous wild animals but also involving introduced domestic animals. This is the largest and most important category for studies of wildlife–domestic animal disease interrelationships. Good examples are African swine fever (Plowright et al. 1969) and African horse sickness (Davies and Otieno 1977), which have reservoirs in wild pigs and zebras, respectively; other examples are canine ehrlichiosis, with its reservoir in jackals (Price and Karstad 1980); trypanosomiasis transmitted by tsetse flies; and malignant catarrhal fever carried by wildebeest. Category 3 diseases have had a long time to evolve a good host-parasite relationship in their wild animal hosts. Characteristically, the wild animals suffer little, if at all; yet they serve as carriers and reservoirs of infections that cause serious diseases in domestic animals.

(4) Infections and parasites of African wildlife which do not involve domestic animals. Examples are a *Theileria* found in impala (Grootenhuis et al. 1975) and the *Taenia* spp. tapeworms of certain wild carnivores which are not transmissible to cattle (Gathuma 1973). The infective agents in this category generally have evolved a temperate or amenable relationship with their wild animal hosts, and because they do not cause disease in our domestic animals we will not consider them further.

Although we have been engaged in research on the disease interrelationships between livestock and wildlife for several years and have also made some observations on the role of wild animals in the epidemiology of several human diseases, much remains to be learned. For example, in collaboration with Mas Bakal of the University of Leiden, we have found a high prevalence (about 80%) of antibodies to *Toxoplasma gondii* in the serum of many species of wild mammals in Kenya (Mas Bakal et al. 1980). What does this mean? Further research is required. Leishmaniasis and tick typhus are examples of human diseases that probably have wildlife reservoirs. Research is indicated. Anaplasmosis is a tick-borne disease of livestock. Many species of wild ruminants have *Anaplasma*-like bodies in their red blood cells (Lohr and Meyer 1973). Are they *Anaplasma marginale* bodies or are these other *Anaplasma* species that are not pathogenic to cattle? One could cite many other questions that demand research.

On the experimental side the project has established captive breeding herds of eland, buffalo, waterbuck, bushbuck, wildebeest, and oryx. Since the immunological status of captured animals is not always easy to determine, these animals are being bred

in captivity and the offspring used for experiments to study pathogenicity and transmissibility of the diseases.

Some of the research on these and other subjects and on certain important zoonotic diseases, such as rabies, echinococcosis, and cysticercosis, are presented in later papers. These contributions report the scientific findings and relate their significance in livestock development and wildlife management. Most but not all of the studies to be described have been carried out in Kenya. There are also papers that look at research as a whole and examine how it relates to different development goals.

In general, the risks of disease transmission between wild and domestic animals have been overestimated and overstressed. Our research has turned up more negative than positive information about such transmission. This has been good for wildlife conservation. We can often tell the livestock producer that the risks of a certain disease, say East Coast Fever, spreading from eland or waterbuck to his cattle are negligible and that the wild animals should be allowed to remain. Such findings and recommendations have removed the early apprehension of wildlife conservationists about our work and have brought the views of wildlife managers and veterinarians into more general agreement, at least as regards diseases. The stage is now set for farmers and ranchers to be given financial incentives to allow game animals to share their lands with their livestock, thereby ensuring the continued existence of large numbers of plains game animals in the face of intensified production of livestock and crops. Wildlife can begin to "pay their way" as a resource that is as valuable to the individual landowner as it is to the Government and at the same time can be cherished and protected.

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The Role of Wildlife in the Epidemiology of Foot-and-Mouth Disease in Kenya

E. C. Anderson[1]

Foot-and-mouth disease (FMD) is a disease of cloven-footed animals, both domestic and wild, caused by a virus of the picornavirus group and characterized by fever and vesicle and ulcer formation in the mouth, on the feet, and on the udder and teats of females. Recovered animals may harbour the virus in the upper respiratory tract for variable periods of time depending on the species.

In considering the possible role of wildlife species in the epidemiology of FMD it is necessary to examine briefly current knowledge on the pathogenesis of the disease in the domestic animal.

Until the late 1950s, it was generally thought that the method by which cattle became infected was by ingestion — the virus entering the bloodstream through lesions in the alimentary tract. However, this concept was revised when infection by airborne virus was shown (Hyslop 1975; Eskildsen 1969). It is now agreed that natural infection takes place via the respiratory tract (Sellers 1971; McVicar 1977) through the inhalation of virus-containing aerosols. In the upper respiratory tract, the pharynx is a predilection site for virus multiplication, and it is in the pharynx that the virus may persist for long periods (Van Bekkum et al. 1959; Burrows 1966; Sutmoller and Gaggero 1965).

Following infection, virus is shed in greatest amounts during the clinical phase of the disease when lesions are present. At this time, aerosols are generated from virus released from ruptured lesions in the mouth and on the feet, teats, and udder and from virus multiplying in the pharynx. Milk and feces from newly infected animals also contain virus.

The exposure of apparently insusceptible animals or immune animals to airborne virus can also lead to infection of the upper respiratory tract. These animals do not develop clinical disease but may excrete virus for up to 7 days following exposure (Sellers et al. 1977; Donaldson 1979). The amount of exhaled virus is proportional to the amount present in the pharynx but is much less than found in clinically diseased animals.

To assess the possible role of wildlife in the persistence and dissemination of FMD the following questions must be answered: (1) What species are susceptible to infection? (2) Do they develop clinical disease and, therefore, generate large amounts of virus? (3) Which species become virus carriers following exposure? (4) Can virus carriers transmit the disease to domestic animals?

Susceptibility of Wildlife Species

An initial assessment of which species might be susceptible to FMD was made by carrying out an extensive field survey for the presence of serum antibody to the five serotypes of virus found in Kenya. Attempts to isolate virus from the throats of many of the animals were also made.

From the results of this survey (Table 1) four species, impala (*Aepyceros melampus*), wildebeest (*Connochaetes taurinus*), eland (*Taurotragas oryx*), and buffalo (*Syncerus caffer*), were selected for controlled laboratory exposure experiments. The method of exposure was either by tongue inoculation (impala, wildebeest, eland, and buffalo) or by nasal instillation (buffalo). The course of the clinical disease, the sites of multiplication, the routes of excretion of virus, and the development of the carrier state were examined (Anderson et al. 1975, 1979, 1980).

Transmission of Disease from Wildlife to Domestic Animals

Eland were found to harbour the virus in the throat for up to 32 days after exposure while buffalo remain carriers for at least 2 years and probably much

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Table 1. Incidence of FMD virus in esophageal–pharyngeal samples and serum neutralizing antibody in wildlife in Kenya.

Species	Virus isolation		Serology					
	Number of samples	Percentage positive (type)	Number of samples	Percentage positive (titre 1.35)				
				O	A	C	1	2
Buffalo (*Syncerus caffer*)	110	13.6(1&2)	135	33	17	7	26	52
Eland (*Taurotragus oryx*)	59	—	79	1.3	—	—	—	11
Grant's gazelle (*Gazella granti*)	13	—	67	6	1.5	—	—	—
Thomson's gazelle (*Gazella thomsonii*)	370	—	481	0.4	0.2	0.4	—	—
Wildebeest (*Connochaetes taurinus*)	170	—	197	—	0.5	—	0.5	0.5
Impala (*Aepyceros melampus*)	289	—	289	—	0.4	—	—	—
Topi (*Damaliscus korrigum*)	—	—	38	0.3	—	—	—	
Coke's hartebeest (*Alcelaphus cokei*)	18	—	52	—	—	—	—	—
Warthog (*Phacochoerus aethiopicus*)	2	—	11	—	—	—	—	—
Waterbuck (*Kobus* spp.)	17	—	27	—	—	—	—	—
Giraffe (*Giraffa camelopardalis*)	2	—	38	—	—	—	—	—
Oryx (*Oryx beisa callotis*)	—	—	11	—	—	—	—	—
Reedbuck (*Redunca redunca*)	—	—	16	—	—	—	—	—
Oribi (*Ourebia ourebi*)	—	—	2	—	—	—	—	—
Dik-dik (*Rhynchotragus kirki*)	—	—	9	—	—	—	—	—
Bushbuck (*Tragelaphus scriptus*)	—	—	4	—	—	—	—	—
Elephant (*Loxodonta africana*)	—	—	177	—	—	—	—	—

longer. Experiments were carried out where eland and buffalo were housed in close contact with susceptible cattle to see if virus transmission would occur.

Results of the field survey for the presence of virus and serum antibody are shown in Table 1. The highest incidence of antibody was found in the buffalo and antibody was found to all five serotypes. Antibody was also observed in a small proportion of eland, Grant's gazelle, Thomson's gazelle, wildebeest, impala, and topi. Virus (types Sat 1 and Sat 2) was isolated from 14% of the buffalo sampled but not from any other species.

Following exposure to virus in the laboratory typical clinical disease was not observed in any of the four species examined. The impala and wildebeest did not harbour the virus for longer than 7 days and the eland for 32 days, but at no time was the virus present in high titre.

In contrast, the carrier state was readily established in the buffalo using the Sat serotypes, and the virus was found in high titre in the throat for up to 3 months after exposure. The buffalo appeared to be less susceptible to type A although the carrier state with this serotype could be established. The buffalo remained carriers for at least 280 days following laboratory exposure and for at least 2 years following natural exposure.

The results indicate that the buffalo is the only wildlife species in Kenya so far recognized as likely to be involved in the persistence and transmission of FMD. Although clinical disease has been reported (Young et al. 1972) it has not been observed in Kenya, Botswana, or Zimbabwe (Hedger 1972; Condy and Hedger 1974; Falconer and Child 1975) and must be considered to be unusual. Consequently the buffalo is unlikely to be a major source of virus, but it will be a persistent source. This has been demonstrated by the isolation of Sat 3 in buffalo in Zimbabwe 15 years after the last recorded case attributable to this serotype in cattle (Hedger 1976).

One must therefore consider if the carrier buffalo can transmit the disease to cattle. In an attempt to answer this question a Sat 2 carrier buffalo was housed with two susceptible grade cattle in a loose-box measuring 6 × 4 m for a period of 25 weeks while a Sat 1 carrier was housed with two susceptible cattle for 5 months.

There was no evidence of transmission in either case. These results must be interpreted with caution as similar attempts at cattle-to-cattle transmission with cattle carriers have also failed. There is some circumstantial evidence of transmission of virus from buffalo to cattle in the field (Condy 1971) as there is of cattle-to-cattle transmission (Anderson et al. 1978).

Buffalo are numerous and widespread throughout Kenya and the incidence of carrier buffalo is undoubtedly high. The virus of FMD circulates readily within the buffalo population because of their gregarious habits. For them to transmit disease to domestic animals, close contact between the species would be necessary to allow airborne transmission to occur.

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Queries about Rinderpest in African Wild Animals

A. Provost[1]

For many years, veterinarians have blamed wild ungulates as being active participants in the rinderpest scene. There is no need to cite references and experimental work on this subject.

In this context, it is puzzling that the veterinary authorities of the African continent embarked in the 1960s in a pan-African rinderpest campaign, known under the name JP_{15}. If wild artiodactyls were a reservoir of virus, there was no hope of eradicating rinderpest from Africa because the prospect of vaccinating the wild fauna was (and still is) rather remote. Despite this evidence, the vaccination campaign has been conducted with success. At present, rinderpest has almost disappeared from Africa, with the exception of the following countries: in West Africa, Mauritania and Mali; and in East Africa, Ethiopia, from where infection spreads from time to time to eastern Sudan and northern Somalia (although the situation is not quite clear in the latter country). Some countries still continue to vaccinate the young stock, others do not vaccinate at all (Guinea, Sierra Leone, Ghana, Togo, Benin, Central African Republic, and Chad). Nevertheless, with the exception of the states already mentioned, no records of rinderpest have been made since 1972 in coastal states and since 1974 in Nigeria. In East Africa, Tanzania, Uganda, and Kenya are reputed to be free of the disease, apart from the scientifically interesting but potentially disastrous isolation of rinderpest virus from an eland found dead in 1974 in the vicinity of Kinna, Kenya, north of Meru National Park.

The disappearance of any clinical evidence of rinderpest from domestic ungulates (cattle, sheep, and goats) has been paralleled by its spontaneous disappearance from wild ungulates. Also, rather interestingly, the few serological surveys undertaken in wildlife have evidenced a sharp decline in terms of numbers positive, with the exception of 4 out of 26 buffalo sera from the Narok district, in southwestern Kenya, which were found positive (FAO 1978).

The discrepancy between this result and the epizootiological evidence is worth further comment. There are only two possible explanations.

Natural Occurrence of a Low-Virulent (Hypovirulent) Strain of Rinderpest Virus

Such strains do exist naturally. It has long been recorded that the passage of a fully virulent rinderpest isolate through a wild species (antelope, giraffe, wild buffalo, wild pig) at first decreases its pathogenicity for cattle and wild animals, although further passages in susceptible domestic calves allow recovery of full virulence. The Masai are aware of this phenomenon and deliberately mingle their cattle with sick buffalo to get "vaccinated" (Scott 1970).

Strains of rinderpest virus already "hypovirulent" for cattle have been isolated on numerous occasions from wildlife in western and eastern Africa. One of the tragic issues is that these seemingly attenuated strains can regain their virulence when they are passaged in cattle.

If such a strain prevails in the buffalo population of Kenya, which could explain the positive results with the buffalo sera, a genuine danger exists, and it may be prophesied that a rinderpest outbreak in cattle will occur because it is unlikely that all weaned calves are vaccinated.

Natural Contact of Buffalo with PPR Virus

The disease "Peste des petits ruminants" is prevalent not only in western and central Africa, but also in the Arabian peninsula, and possibly in India. Surprisingly enough, it has not been recorded from

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eastern Africa although I have a strong feeling that it does exist in Sudan where it may have been confused with rinderpest. In the laboratory, confusion is possible because these two "brother" viruses have almost identical behaviours in tissue culture, gel diffusion, and other serological tests. The difference lies in the pathogenicity for cattle: PPR virus is apathogenic for cattle although infective (with subsequent immunity to rinderpest); rinderpest is pathogenic. Another, more subtle difference, is the disparity of magnitude in serum-neutralization tests when they are performed with either the homologous or the heterologous virus: sheep and goat sera react with higher titres when tested with PPR virus than when tested with rinderpest virus and reversely, cattle sera (from rinderpest immune animals) have lower titres to PPR virus than to rinderpest virus.

Now comes a crucial question: In nature, is PPR virus able to infect other ungulates spontaneously, exclusive of any clinically recognizable reaction? I think that this question cannot be answered, possibly because it has never been raised.

I have a feeling this question may be answered by looking at very recent findings in Oman. In that country, where rinderpest is unknown in human memory,[2] it has been found that small ruminants have genuine PPR antibodies but also that unvaccinated cattle have higher antibody titres to PPR than to rinderpest virus.

Is it possible that this is the case in Kenya for buffalo? The approach is then simple and lies in the performance of comparative titrations of PPR and rinderpest antibodies on the same sera.

I hope that the results of the survey are positive, because it would be less alarming, and certainly more scientifically fruitful, to assume the possible occurrence of PPR infection in the buffalo population rather than the existence of a hypovirulent strain of rinderpest virus. I would also urge that the eland isolate from 1974 be reexamined in the light of these comments.

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[2]The survey was undertaken before a virgin outbreak of rinderpest flared up in the country, introduced from a neighbouring state.

Epidemiology and Control of Bovine Malignant Catarrhal Fever

E. Z. Mushi,[1] F. R. Rurangirwa,[1] and L. Karstad[2]

Malignant catarrhal fever (MCF) is a clinicopathological condition of cattle that is characterized by fever, ocular and nasal mucopurulent discharges, bilateral corneal opacity, and generalized lymph-node enlargement.

The disease has a worldwide distribution and two forms of the disease have been recognized. "Wildebeest-derived" sometimes called "African MCF" occurs when cattle and wildebeest graze together. A herpesvirus has been identified as the causative agent. This virus is carried latently by wildebeest (Plowright et al. 1960). The second type is "sheep-associated" or "European MCF," which occurs in areas without wildebeest. The causative agent of "sheep-associated" MCF has not yet been isolated and because this type of MCF is not a major problem in Kenya only the wildebeest-derived MCF will be discussed.

In Masailand, where cattle and wildebeest share grazing land, a morbidity rate of 7% has been attributed to MCF virus infection and because all infected cattle die, the disease constitutes an economic problem. Besides the 100% mortality, the wildebeest compete with cattle for the available pastures. In fact, the Masai have asked the government to control or eliminate the wildebeest from their grazing land. The confinement of wildebeest in National Parks with the help of game-proof fences would reduce the problem but the erection of game-proof fences around all parks is both too expensive and impractical.

We report on the epidemiology and on attempts at immunization against wildebeest-derived MCFV as an alternative method of control of this disease.

Shedding of MCFV by Wildebeest

Although MCF has been associated with the close grazing of cattle and wildebeest, the exact mode of transfer of infection remains unknown (Mettam 1923; Plowright 1968). However, transmission by pen contact between wildebeest calves and cattle has been demonstrated (Plowright 1964, 1965).

The Masai believe that MCF is contracted when cattle graze on pastures contaminated by the afterbirth of wildebeest or on grounds where wildebeest calves have shed their natal hair coats (Daubney and Hudson 1936). Attempts to isolate virus from wildebeest placenta or hair have been unsuccessful (Mushi, unpublished).

Wildebeest secretions and excretions were tested for viral infectivity in calf thyroid cell cultures. MCFV was isolated from nasal and ocular secretions of young free-ranging wildebeest calves from the Athi-Kapiti Plains (Mushi et al. 1980a). Virus was isolated from wildebeest calves that were from a few days old to less than 3 months of age (Table 1). The isolation of MCFV from wildebeest calves only a few days old would suggest that the calves acquire the infection in utero.

In contrast to the cytomegaloviruses that are regularly shed in saliva and urine (Utz 1964), MCFV was not isolated from wildebeest calf saliva or urine.

The virus in nasal and ocular secretions occurs in a cell-free state, which is easier to transfer from animal to animal. Cattle also shed MCFV in their nasal and ocular secretions, but in cattle, all infectivity was found to be cell-associated (Mushi and Rurangirwa 1980). The inability of MCFV to spread by contact in cattle is probably due to the absence of cell-free virus in these secretions.

The transmission of MCFV from wildebeest calves to cattle is most likely by contamination with infected secretions. Wildebeest generate an aerosol during their habitual "snorting." Contamination of drinking water and pastures is also a possibility. The infection also could be mechanically carried by flies from wildebeest calves' eyes and nostrils to the same points in cattle.

MCFV has been isolated from tissues of corneas and turbinates of young wildebeest calves (Mushi et al. 1980a). This would suggest that the virus replicates in these sites. Probably the virus replicates

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Table 1. MCF virus isolation from wildebeest-calf secretions and excretions.

Virus titre (log_{10} $TCID_{50}$/ml)				Serum VN antibodies[a]
Nasal	Ocular	Saliva	Urine	
2-month-old animals				
—[b]	NT[c]	—	—	1.35
3.2	NT	—	—	1.28
—	NT	—	—	0.50
NT	3.2	—	—	0.98
1.7	Trace	—	—	1.20
2.5	2.6	—	—	1.80
3-month-old animals				
2.6	—	—	—	1.43
—	—	—	—	1.80
—	—	—	—	1.50
—	—	—	—	2.18
—	0.7	—	—	1.50
—	—	—	—	1.65

[a] Equals log_{10} VN_{50}.
[b] Nil.
[c] Not tested.

on the corneal epithelium and passes down the nasal/lacrimal duct to the nasal chamber as is the case with IBR virus (McKercher et al. 1963).

MCFV was not isolated from wildebeest calves more than 3 months old. However, a virus-neutralizing antibody has been demonstrated in calves over 3 months of age (Mushi et al. 1980b). Virus-specific IgA with neutralizing activity has been demonstrated in nasal secretions (Rurangirwa et al. 1980). The cessation of virus shedding coincides with the appearance of virus neutralizing antibodies in nasal secretions and this may explain the seasonal occurrence of MCF.

Separation of cattle from wildebeest during the calving season and for the following 3 months would reduce the risk of cattle contracting MCFV infection.

Immunization

Studies on the immunity of MCFV infection have been greatly hindered by the fact that the disease causes very high mortality; however, the few cattle that do survive infection acquire a solid resistance (Plowright 1968). Because this disease causes high mortality, the demand for an immunizing agent has been high, and several unsuccessful attempts to develop a vaccine have been made.

Inactivated vaccines consisting of formalinized infected cattle lymph-node suspensions failed to protect cattle or rabbits against virus challenge by inoculation (Piercy 1954). Inactivated cell-culture virus induced high and persistent levels of virus-neutralizing antibodies in cattle but no protection against infection. Recently Edington and Plowright (1980) showed that formalin-inactivated MCFV, with Freund's complete adjuvant, was able to protect rabbits against two consecutive parenteral challenges with cell-free virus but some "protected" rabbits succumbed to challenge with cell-associated MCFV.

Several attempts to attenuate the herpesvirus of MCF have been unsuccessful. Repeated passage of the virus in cattle, rabbits, or tissue culture produced virus with diminished virulence but not attenuated for cattle (Plowright 1968).

Another approach to the immuno-prophylaxis of MCF is the use of naturally avirulent strains of MCFV to immunize cattle. Because antibodies to MCFV have been demonstrated in three species of Alcelaphinae (wildebeest, hartebeest, and topi) and one species of the Hippotraginae (the oryx), these animals have been tested for MCF-like viruses.

A herpesvirus isolated from a hartebeest did not consistently protect cattle against parenteral challenge with wildebeest-derived virus (Reid and Rowe 1973).

A herpesvirus isolated from topi did not induce disease in cattle and rabbits. This virus was shown to be antigenically related to the wildebeest-derived MCFV, but cattle inoculated with cell-associated topi virus did not withstand challenge with cell-associated wildebeest-derived MCFV. Nevertheless, the protective effect of this topi virus, particularly when cell-free, should be further investigated.

Also, the oryx should be investigated for the presence of an avirulent strain of MCFV because antibodies to MCFV have been demonstrated in these

animals but no virus has yet been isolated from them.

At present the only practical way to control MCF is to remove cattle from pastures frequented by wildebeest during the calving season and for the following 3 months.

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The Possible Role of Wildlife as Maintenance Hosts for Some African Insect-Borne Virus Diseases

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The coastal strip of Kenya has been inhabited for many years, while it is only over the last 400 years that the highland areas have been populated by the movement of peoples from the south and the north. The natural fauna, the wildlife inhabiting the forest and bushed grasslands, now share their habitat with the domestic ruminants brought by the Nilotic and Bantu peoples. Many insect-borne viruses had established maintenance cycles in the forest and savanna habitat. These involved the natural fauna of the region and showed little or no evidence of disease. The coming of the domestic stock several centuries ago and the importation of more exotic species this century introduced many new potential disease hosts, as did the coming of the people themselves. The impact of these intrusions has become more and more interesting and relevant to the development of the region, both for animal production and for the health of the human population.

Diseases such as bluetongue in the wool sheep, Nairobi sheep disease, Rift Valley fever, ephemeral fever, and lumpy skin disease were recognized in Kenya with the development of the agricultural industry. African horse sickness appeared in horses brought into the territory. The causative viruses were suspected to be transmitted by insects and the elucidation of the natural maintenance cycles for these viruses is a subject of considerable interest and relevance to their ultimate control.

At Kabete, investigations were carried out on the distribution of diseases, and of antibodies and vectors where these were known, in relation to the ecological zones in Kenya. The antibody surveys were carried out both in the disease host populations and also among the natural fauna of the region. Some of the diseases initially behaved in an epizootic manner and later contracted to persist in enzootic maintenance cyles in limited habitats. Fundamental studies were carried out, in particular with *Culicoides* (Walker 1976), to determine their distribution, population biology, feeding and breeding habits, and the foraging ranges. Attempts were also made to isolate viruses.

Investigations were made of the role of wildlife in these diseases. Initially, efforts were made to isolate viruses from wild game animals killed for control purposes; more than 100 samples were inoculated into sheep, BHK cell cultures, infant mice, and embryonated eggs. Two passages were made in the latter three systems. This work did not result in any virus isolations (Davies 1980). The serological investigations generally were carried out with one test but the results were always validated by a further serological test for the same disease.

Bluetongue

Sera from a wide range of wild ruminant species collected in many different ecological zones contained both fluorescent and type-specific neutralizing antibody to bluetongue virus. Animals such as the buffalo (*Syncerus caffer*), wildebeest (*Connochaetes taurinus*), kongoni (*Alcelaphus buselaphus cokei*), and various gazelles (*Gazella* spp.) are probably important maintenance hosts for bluetongue virus in Kenya and many other parts of Africa. Large proportions of their populations have shown evidence of challenge by bluetongue virus (Davies and Walker 1974; Davies, unpublished). In the absence of cattle, which have replaced the wild ruminants in many developed parts of the country, it is considered that they play the major role in the virus maintenance cycle. Blood meal analysis of the species of *Culicoides*, which are considered important vectors of bluetongue, show that they feed principally upon wild and domestic ruminants (Walker and Boreham 1976). Where agricultural development has largely excluded wild species, domestic cattle assume the principal role in the maintenance

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of the virus (Davies 1978). There are large parts of Africa where agricultural developments in the next 50 years may introduce disease hosts. The bluetongue virus maintenance cycle probably already exists in those countries.

Ephemeral Fever

Ephemeral fever occurs in Kenya in the form of epidemics which sweep across large parts of the country (Davies et al. 1975). Between the epidemics, limited foci of disease may be encountered and there is evidence of seroconversions in sentinel herds. An examination of a range of wild ruminant sera showed evidence of neutralizing antibody in 54% of buffalo, 62% of waterbuck, 9% of wildebeest, and 2.8% of hartebeest. These samples had been collected from the higher potential agricultural areas of the country through to the more arid acacia savanna grasslands. Antibody is also found in the domestic cattle population in these zones. A further interesting observation was that there were seroconversions in waterbuck and buffalo that were not alive during the previous epizootic of the disease. This is evidence that the virus was cycling in the wild ruminant population during a period when no clinical disease was diagnosed in cattle (Davies et al. 1975). These animals may be important maintenance hosts for this virus in Kenya.

African Horse Sickness (AHS)

The only Equidae in Kenya before the importation of horses were the zebras (*Equus* spp.). As soon as horses were imported into the country in this century, large numbers died of the disease. This is a classic situation of a virus finding a disease host. No disease has ever been recognized in the wild zebra population, and attempts to isolate the virus from zebra blood have proved unsuccessful. Antibody has been found in zebra sera by complement fixation, fluorescent antibody, and neutralization tests (Davies and Lund 1974; Davies and Otieno 1977; Davies, unpublished).

As a follow-up of some apparently low-titre neutralizing antibody titres in elephant sera, a number were screened by a complement-fixation test (Davies and Otieno 1977). Some 84% were found to contain complement-fixing antibody to AHS antigen. The significance of this is not understood and it may be due to another virus closely related to AHS. There is little doubt, however, that wild zebra play an important role in the maintenance and amplification of AHS virus in East Africa.

Rift Valley Fever

This is one of the most pathogenic zoonoses known and was first identified in the Rift Valley in Kenya in 1931 (Daubney et al. 1931). The disease principally affects cattle, sheep, and goats in an epizootic manner at intervals of up to 10 years. Man is accidentally infected, generally by contact with the diseased animals. A survey of wild ruminant sera collected after an epizootic in Kenya in 1968 showed that there was very little involvement of the wild ruminant populations. A very small and insignificant proportion of wildebeest and hartebeest were found to contain antibody. This was not surprising, for the game animals generally inhabit areas which are outside the epizootic range of the disease (Davies 1975a). Buffalo infected experimentally with the virus developed a viraemia and one of four animals aborted in a manner similar to that expected in cattle (Davies and Karstad 1980).

Between epizootics, the virus maintenance cycle contracts into certain forest regions of Kenya. It is not clearly known what vertebrate is involved in the maintenance cycle. Cattle are involved to a certain extent where they graze at the forest edge (Davies 1975a) and certain wild ruminant species in the forest may also be involved. Buffalo are likely hosts in such areas, but it has not been possible to confirm this (Davies and Karstad 1980). Further investigations have included baboons (*Papio anubis*) (Davies et al. 1972), green monkeys (*Cercopithecus aethiops*) (Davies and Onyango 1978), birds (Davies and Addy 1979), and rodents. No evidence for the involvement of these species has been obtained. The problem of defining the interepizootic vertebrate hosts remains.

Lumpy Skin Disease

There is no direct evidence to show that the virus is transmitted by a biting insect, however, the circumstantial evidence strongly supports this view (Diesel 1949; MacOwan 1959). The capripox viruses have cross relationships with one another and epidemiological investigations using serological tests suffer the disadvantage that any antibody detected may be due to a sheep and goat pox virus, or at low titres to cowpox. The latter however does not occur naturally in Kenya. There is no evidence of any infection of the wildlife populations in areas where epidemics of sheep and goat pox have occurred (Davies 1975b).

Lumpy skin disease was first recognized in Kenya in 1957 when it appeared in epizootic form, although with a much lower morbidity than was experienced

in South Africa. Since that time, the disease has occurred sporadically in most years with only one or two clinical cases recognized. These have always been in certain high altitude districts adjacent to a large forest area and frequently in farms excised from the forest. Because the capripox viruses have a very narrow host range, it was thought likely that the wildlife maintenance hosts must exist in the larger wild ruminants (e.g. buffalo) found in such habitats. In sera from such ecological zones and those contiguous with them, neutralizing antibody was found in a high proportion of the sera examined (Davies, unpublished). Buffalo sera from areas where the disease has not occurred did not contain antibody. In samples from Uganda taken in a year after an epizootic of the disease, a high proportion contained neutralizing antibody to lumpy skin virus. None of the 166 buffalo sera contained antibody to a strain of cowpox virus. If the antibody detected in the buffalo sera is in fact specific, these animals could be important reservoirs for the disease.

Nairobi Sheep Disease

This is a tick-borne disease of sheep and goats transmitted principally in Kenya by *Rhipicephalus appendiculatus* (Montgomery 1917; Davies 1979a). This tick feeds largely upon cattle and the larger wild ruminants. Smaller proportions feed upon sheep and goats but generally they are less preferred feeding hosts than bovids. A survey of wildlife sera, collected in areas where this tick was common, showed few with any antibody to the virus, and then only at a very low titre. These low titres were confirmed by complement fixation, fluorescent antibody, and indirect hemagglutination tests (Davies 1979b) and it was considered likely that they were cross reactions with other viruses. This suspicion was given support by the finding that Nairobi sheep disease was probably identical with a virus, Ganjam, from India and reacted on fluorescent antibody tests with Congo, Dugbe, and some other tick-borne viruses (Davies et al. 1978). There are reports of antibody to Congo and Dugbe viruses in wild ruminant sera detected by agar gel precipitation.

The conclusion from this work is that wild game animals do not play any role in the maintenance of this virus, supporting a hypothesis that it is an introduced virus. Sheep and goats are not indigenous to East Africa but may have been brought from India to the coast years ago. The noninvolvement of game animals in sheep and goat pox indicates that this also may have been introduced. The remainder, bluetongue, ephemeral fever, African horse sickness, and lumpy skin disease, are likely to be indigenous to the East African ecosystems and be maintained in the wild game populations of the region. Rift Valley fever also is an indigenous virus, utilizing the forest habitat for maintenance and probably some wild vertebrate species.

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The Possible Role of Wildlife in the Natural History of Rabies in Kenya

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Rabies has been known in Kenya for many years and originally was widespread over much of the country. Early reports indicate that many cases were encountered in silver-backed jackals and dogs and that these were considered to be important in the propagation of the virus. Vigorous control of animal movements and the shooting and poisoning of wild carnivores and stray dogs gradually restricted the range of the disease. By 1967, the whole of western Kenya was free from the disease, but sporadic cases were found in the large Northern Province, and rabies remained a problem in the relatively heavily populated parts of Eastern Province in Kitui and Machakos districts. Despite rigorous control measures, including the vaccination of dogs, the shooting and poisoning of all strays and some wild carnivores, cases have occurred every year with no decrease in numbers.

In the 10 years to 1967, 10 silver-backed jackals, 2 white-tailed mongooses, 2 bat-eared foxes, 1 hyena, and 1 civet cat were confirmed to have died of rabies in these areas. The results from the period 1968–79 are shown in Table 1. The period 1967–76 represents an unaltering situation with a few cases occurring every year in these two districts, in dogs, other domestic animals, and a few wild animals. The number of cases that arrive at the laboratory for confirmation is probably a small proportion of those that actually occur.

The range of the wild animals that are involved is not restricted to the zones where the disease occurs; it extends over much wider areas. The range includes the much more sparsely populated game parks or pastoralists' range country where the likelihood of contacts of the wild animals with the indicator of disease, the domestic dog, is reduced. The disease has not spread the 40 miles (65 km) to the urban centre of Nairobi. This has always been a point of interest. What is the ecological barrier? It is considered that the persistent enzootic area in Machakos and Kitui reflects the population density and that the persistence of the disease in the face of the control measures that have been successful in other parts of the country makes it likely that there is a sylvatic cycle for the maintenance of the virus. The table shows the likely candidates for this role: the honey badger (*Mellivora capensis*), the white-tailed mongoose (*Herpestes* sp.), and the silver-backed jackal (*Canis mesomelas*). In 3 months, one field officer submitted 6 honey badgers, all of which were confirmed to have had rabies. An intensive field investigation might reveal which animals are involved in this maintenance cycle.

In late 1976, rabies appeared in dogs in the coast hinterland. The disease was thought to have been introduced from northern Tanzania where there were many cases at that time. The epizootic moved rapidly in the semi-urban dog population northwards up the Kenya coast and covered some 150 miles (240 km) in 1 year.

Vaccination and the shooting and poisoning of stray dogs gradually brought the epizootic under control. There was no evidence of any wildlife involvement in this outbreak.

In 1979, rabies appeared again in western Kenya after a long absence, with cases in dogs in border areas north and south of Lake Victoria. There had clearly been movement of rabid animals across the borders from Uganda and Tanzania where rabies had not been controlled for some years. Many dogs, domestic and wild animals, and humans were confirmed to have died of the disease. Efforts at control are producing a reduction in the number of clinical cases.

Northern Province

In this arid and semi-desert territory, there is some evidence of the disease occurring in periodic epizootics that may be related to population fluctuations

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Table 1. The various vertebrate hosts confirmed to have died of rabies in Kenya (1968–79).

	Dogs	Cats	Sheep	Goats	Cattle	Donkey	Jackal	Mongoose	Civet cat	Honey badger	Hyena
1968	5	1	—	—	2	—	2	—	1	1	—
1969	6	—	—	—	2	—	—	1	—	—	—
1970	0	1	—	1	1	—	—	—	—	—	—
1971	3	—	—	—	4	—	—	1	—	—	—
1972	11	—	—	1	—	—	—	—	—	—	—
1973	3	—	—	1	1	1	—	—	—	2	—
1974	9	2	1	—	3	—	—	—	1	1	—
1975	10	—	—	—	2	—	—	1	—	—	—
1976	60[a]	3	—	2	2	—	—	1	—	1	—
1977	35[b]	1	—	1	—	1	1	—	1	—	1
1978	11[c]	—	—	1	1	—	1	—	1	1	1
1979	14[d]	—	—	—	1	1	1	—	—	6	—

[a] Machakos 12; Coast 48.
[b] Machakos 9; Coast 26.
[c] Machakos 9; Coast 2.
[d] Machakos 8; Coast 6.

of wild animals such as the jackal. Cases have been confirmed in dogs, in some other domestic animals, and in hyenas and jackals close to the population centres over a number of years, but in such a large and sparsely populated area it is impossible to get a true picture of the disease problem.

Conclusions

There are good grounds for suspecting that there is a sylvatic reservoir of rabies in Eastern Province, Kenya. The range of this reservoir may be much greater than that indicated by the occurrence of clinical disease in dogs, which is largely a reflection of the human population density. In northern Kenya there is a suspicion that there may be sylvatic rabies involving the jackal and possibly other carnivores. There is a need to investigate the natural history of rabies in East Africa on a broader basis in the different ecological zones. Intensive study is required in the dry northern territories of Kenya, Ethiopia, and Somalia and in the bushed and wooded grasslands prevalent in much of Kenya, Tanzania, and Uganda.

Attempted Isolation of *Cytocoetes ondiri* from Wild Ruminants in Areas where Bovine Petechial Fever is Endemic

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Bovine petechial fever (BPF) is a hemorrhagic syndrome encountered in both *Bos indicus* and *B. taurus* and found only in Kenya (Piercy 1953; Danskin and Burdin 1963). There is some evidence that it may occur on the slopes of Mount Kilimanjaro in Tanzania, where it is known as "bushbuck disease" by the farmers. The significance of the bushbuck (*Tragelaphus scriptus*) as a reservoir host for the causal agent, *Cytocoetes ondiri* (Krauss et al. 1972) was suggested by the isolation of the agent from the blood of three of five bushbuck killed in an endemic area (Snodgrass et al. 1975). Many other wild ruminants inhabit such areas.

There is very good field and laboratory evidence to show that BPF is transmitted by an arthropod vector, but the identity of this is not known. The disease only occurs in forest or forest edge situations or where there is good ground cover (Walker et al. 1974). Animals need to enter the challenge area to contract the disease, merely grazing alongside where they are fenced does not result in infection. There is thus a distinct analogy with scrub typhus; the nidus of infection is limited in extent. The occurrence of BPF cannot be correlated with any breakdowns in the control of tick-borne disease; on the contrary, it regularly occurs in areas where a high challenge by diseases such as "corridor disease" is effectively controlled by dipping or spraying with acaricides.

This paper further attempts to define the natural host range of BPF, and the ectoparasites found on the principal reservoir host. Animals were killed in areas where the disease had been shown to occur, or where it might be expected to occur on ecological grounds. Unfortunately, certain collections were made after a prolonged dry spell in 1976 when no clinical disease had been seen for some time and where the habitat had been altered by overgrazing in the drought conditions. The animals were shot, and the blood was collected in heparin as soon as possible either by venesection or directly from the heart. Volumes of 20–100 ml were transported to the laboratory on ice, usually within 24 hours of collection. The collection sites were: (1) Ol Mogogo — a known endemic area for BPF on the edge of the Rift Valley; (2) Timau — an area of cedar forest where the disease had been identified in cattle 1 week previously; (3) Aberdares — an area considered to be endemic on ecological grounds but where the disease had not been confirmed; (4) Sugoroi — a likely endemic area where the disease had been reported by farmers, but not confirmed at the laboratory (sampled after a long dry period); and (5) Nderit — an acacia woodland area, where the disease had been confirmed.

Isolation of *Cytocoetes ondiri*

The blood samples were used for the isolation of the organism as described by Snodgrass et al. (1975). The reaction in sheep, which were used for the isolation, was monitored as described by Krauss et al. (1972). In the case of a doubtful reaction, 50 ml of blood from the suspect sheep was subinoculated into another test animal.

Ectoparasites

These were collected from the dead animals and either kept alive in a humidified container or placed directly in alcohol for identification. This was carried out by G. Backhurst.

Results and Discussion

The summary of the isolation attempts is shown in Table 1. There was a total failure to isolate the BPF agent from the series of bushbuck killed at an

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Table 1. The isolation of *Cytocoetes ondiri* from wild ruminants.

Species	Origin	Enzootic area	Year	Number	Number positive
Waterbuck	Nderit	Yes	1970	23	0
	Ol Mogogo	Yes	1976	1	0
	Ngobit	?	1976	1	0
Suni	Sugoroi	?	1976	2	0
Steinbuck	Sugoroi	?	1976	2	0
	Ol Mogogo	Yes	1976	2	0
Grey duiker	Ngobit	?	1976	2	0
Impala	Ol Mogogo	Yes	1974	1	0
	Sugoroi	?	1976	1	0
Mountain reedbuck	Sugoroi	Yes	1975	2	0
		?	1976	2	0
Dik-dik	Ol Mogogo	Yes	1975	1	0
			1976	4	0
Bushbuck	Ol Mogogo	Yes	1975	6	4
			1976	5	0
	Timau	Yes	1976	3	1
	Aberdares	?	1977	3	3
	Ngobit	?	1976	2	0
	Sugoroi	?	1976	9	0

endemic site (Ol Mogogo) in 1976. Previous isolation rates had been three of five positive in 1972–73 and four of six positive in 1975. The 1976 sampling was after a drought and the characteristics of the vegetation at the site had been extensively altered by the grazing pressure. No positives were found at the Sugoroi site in 1976 and the explanation could be the same because it was extremely dry. No isolations were made from any other species. Important negative findings were recorded from the 23 waterbuck, which are a common species in endemic areas. The isolation rates obtained at Ol Mogogo in 1975, Timau, and the Aberdares serve to confirm the levels of involvement of the bushbuck in the natural history of BPF.

The ectoparasites collected from bushbuck were ticks of the family Ixodoidea (Table 2). Two genera, *Haemaphysalis* and *Ixodes* were those most commonly found. Some collections of *Haemaphysalis aciculifer* taken from positive bushbuck were ground up and inoculated into susceptible sheep; no reactions ensued and the sheep remained susceptible to

Table 2. Tick species taken from bushbuck collected in areas known to be, or ecologically likely to be, enzootic for bovine petechial fever.

Bushbuck	BPF isolation	*Haemaphysalis aciculifer*	*H. parmata*	*Hyalomma truncatum*	*Ixodes* spp.	*Rhipicephalus appendiculatus*	*R. evertsi*	*R. hurti/ jeanneli*
1	—	14[a]	—	—	—	2	—	—
2	—	14	—	—	3	1	3	2
3	—	10	—	—	7	1	—	2
4	—	8	—	—	1	2	—	2
5	—	15	—	—	—	6	—	8
6	—	+[b]	+	—	+	+	+	+
7	Positive	2	—	—	68	1	—	—
8	—	5	—	—	39	—	—	—
9	—	2	—	—	51	1	—	—
10	—	7	2	—	12	18	2	1
11	—	26	18	—	33	—	—	—
12	—	—	2	1	5	1	7	7
13	—	3	4	—	28	2	2	2
14	—	3	1	12	13	6	6	10
15	—	6	—	1	—	1	—	—
16–18	Positive	72	49	2	—	—	3	5
19	Positive	68	18	3	—	—	—	2

[a] Number of specimens.
[b] Present.

BPF. Larvae hatching from engorged females collected from positive bushbuck were successfully fed upon susceptible sheep; some 79 engorged specimens were collected. No reactions followed in the sheep, which remained susceptible to BPF challenge.

Many bushbuck carried *Haemaphysalis* or *Ixodes* ticks and were negative for BPF during the dry 1976 collection. This might support a view that vectors other than ticks are responsible for the transmission of BPF. Acaricides have no effect on the natural transmission of infection in challenge areas. All other related organisms such as tick-borne fever are tick transmitted, but positive evidence for the transmission of BPF by ticks is still lacking and there is much indirect evidence to suggest that it is unlikely.

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The Importance of Wildlife in the Epidemiology of Theileriosis

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An important task for scientists involved in research on animal health in Kenya and elsewhere in Africa is to determine the role that the abundant wildlife population plays in the epidemiology of diseases of livestock. Only then can investigations into possible control measures be undertaken. One of the most important cattle diseases in Kenya is East Coast fever, which is caused classically by the protozoan parasite *Theileria parva* and is transmitted by the brown ear tick *Rhipicephalus appendiculatus*. It has been estimated in East Africa alone that half a million cattle die a year from East Coast fever (ECF) (Miller et al. 1977). The only control measure used for ECF at present is the application of acaricides at frequent intervals, which is very time consuming, expensive, and often not effective. Additional effects of ECF are the exclusion of highly productive cattle from endemic areas and the loss of productivity of cattle that recover from the disease. Research on the control of ECF has recently been complicated by the realization that theileriosis of cattle is in fact caused by a collection of *Theileria* species acting individually or together. Because of the importance of ECF as a cattle disease it is essential to determine the extent and importance of the wild Bovidae both for the maintenance of *Theileria* spp. infective to cattle and for the maintenance of the tick vectors of *Theileria*.

There are more than 40 species of wild Bovidae in East Africa and the majority of these populations are harbouring *Theileria* parasites in their erythrocytes (Brocklesby and Vidler 1966; Grootenhuis and Young, unpublished). While some of these *Theileria* parasites have been given specific names, only in the case of *T. lawrencei*, which occurs in the majority of African buffalo within the range of *R. appendiculatus* (Young et al. 1978), has its relationship to cattle theileriosis been established in any detail. Cattle and wildlife have close contact in most areas of East Africa and usually share pastures in pastoral areas and even on developed ranches and farms. *R. appendiculatus* is by far the most important tick species for the transmission of *T. parva*, *T. lawrencei*, and some other *Theileria* species. This tick has a wide host range and can infest most bovid species in East Africa (Yeoman and Walker 1967; Walker 1974). Similarly, ticks of the genus *Amblyomma* which are the main vectors of *T. mutans* and *T. velifera*, have a wide host range on domestic and wild ungulates. Because cattle and wildlife have such close contact, the tick vectors of *Theileria* can be maintained and transferred between domestic and wild Bovidae. This situation can be illustrated by *Amblyomma cohaerens*, which mainly occurs on the African buffalo and only infests cattle in areas of close buffalo/cattle contact such as the Trans Mara (Newson, Young and Moll, unpublished results). Under these circumstances this species is undoubtedly an important vector for the transmission of *T. mutans* between buffalo and cattle (Young et al. 1977a, Young, unpublished).

The objective of this paper is to review the studies already carried out on the role of wildlife in the epidemiology of cattle theileriosis, especially the ongoing collaborative research between the Wildlife Diseases Section, Kabete, and the Veterinary Research Department, Muguga. In order of their established importance in the epidemiology of disease in domestic animals the role of buffalo, eland, and other wild bovid species will be discussed.

Buffalo and Theileriosis

The role of buffalo was first confirmed in South Africa by Neitz (1955) who described "corridor disease" from cattle associated with buffalo. This form of theileriosis was distinct from classical ECF because: (1) infected cattle did not infect ticks, because of the absence of intraerythrocytic piroplasms in the blood of infected cattle; (2) the mortality of cattle

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in the buffalo inhabited "corridor" area ceased when the cattle were moved outside the buffalo zone; (3) only a few macroschizonts of small dimensions were found in the tissue smears of infected cattle; (4) splenectomy caused a recrudescence of piroplasm parasitemia; and (5) a carrier state occurred in buffalo in contrast to *T. parva* infection in cattle. Buffalo appeared normally to be unaffected by this *Theileria* although Neitz reported that two buffalo calves apparently died of this *Theileria* species. Neitz (1955) considered that these characteristics of the buffalo species were enough to separate it from *T. parva* so he created a new species *T. lawrencei*. However, Neitz (1957) reported that cattle that had recovered from *T. lawrencei* had a high degree of protection against *T. parva* challenge.

In the 1960s extensive studies were carried out on *T. lawrencei* from Kenyan buffalo by Barnett and Brocklesby (1959, 1966, 1968). They confirmed many of Neitz's findings and observed a similar epidemiology of *T. lawrencei* in Kenya. In experimental infections of captive buffalo calves, Brocklesby (1964) found that calves became infected and that one died of *T. lawrencei* compared with the development of transient *T. parva* infection in two of six buffalo. The parasite could be transmitted to cattle from only one buffalo. They showed that *T. lawrencei* when it could be passaged by ticks through cattle changed its behaviour so that it eventually produced a disease syndrome identical to ECF. These results plus the fact that *T. lawrencei* recovered cattle were resistant to *T. parva* challenge led Barnett and Brocklesby to suggest that *T. lawrencei* was merely a biological variant of *T. parva*. Using improved techniques, Young et al. (1973), Young and Purnell (1973), and Young, (unpublished) confirmed the transformation of *T. lawrencei* from Tanzanian buffalo after tick passage in cattle. Young and colleagues showed that cattle-adapted *T. lawrencei* remained highly pathogenic to cattle while Brocklesby and Barnett reported a reduced pathogenicity. It is suggested that *T. lawrencei* from buffalo does not normally complete its life cycle in cattle, but the few parasites that do complete their cycle can be selected during further cattle passage. Young et al. (1978) reported very high prevalence of *T. lawrencei* in buffalo in East Africa within the distribution of *R. appendiculatus*.

Immunization of Cattle

Immunization against *T. lawrencei* by infection and treatment and cell culture methods have been described (Young et al. 1978). However, several problems arose during a series of cross immunity experiments. Young et al. (1973), and Radley et al. (1975, 1979) found that *T. lawrencei* immunized cattle were generally resistant to challenge with *T. parva*, but *T. parva* immunes died on challenge with *T. lawrencei* from buffalo. In addition, several *T. parva* isolates were not found to be cross-protective in similar experiments. In another series of experiments Young et al. (1977a,b) found evidence of antigenic variation in *T. lawrencei* carrier buffalo. The existence of heterogenous immunogenic populations of *T. parva* and *T. lawrencei* is at present the greatest barrier in the immunological control of ECF. Effective protection against cattle-maintained disease may be possible, but strong evidence is mounting that buffalo-derived disease may be much more difficult to protect cattle against. At present, the extent of the antigenic repertoire of *T. parva* and *T. lawrencei* is largely unknown. It appears that the *T. lawrencei* of buffalo has the most extensive repertoire and this animal is therefore a key species for further studies on immunological control of ECF. Recent studies by Dolan and colleagues at Muguga have shown that drugs effective for the treatment of *T. lawrencei* may be forthcoming.

At present, the Wildlife Diseases Section, Kabete, is collaborating with the Veterinary Research Department, Muguga. A small herd of breeding buffalo has been established to obtain *Theileria*-free buffalo because nearly all buffalo in the wild have been found to be *Theileria* carriers (Young et al. 1978).

Studies with *T. lawrencei*

Many of the buffalo used in the studies reported above were carrying *Theileria* parasites so they were unsuitable for experimental infections. For any experimental work with buffalo, *Theileria*-free animals must be used.

One of the more interesting questions is what would happen if a *T. lawrencei* strain adapted to cattle could be passaged through buffalo. The first attempt to infect cattle with a cattle-adapted or "transformed" strain of *T. lawrencei* was complicated because the buffalo was later shown to be a *T. lawrencei* carrier before infection. This experiment still produced some interesting results. Three tick isolations were obtained from this buffalo, one before experimental infection with *T. lawrencei* (Serengeti transformed), adapted to cattle by six serial passages, and two further isolations after inoculation of this stabilate. The first and third isolations behaved in cattle like classical *T. lawrencei* from buffalo but the second isolation behaved like

T. parva. These results suggest that "transformed" *T. lawrencei* infected the buffalo and was not inhibited by the carrier state of the "non-transformed" *T. lawrencei*. It also gave credence to the hypothesis that buffalo can harbour more than one strain of *T. lawrencei* at one time (Young et al. 1978). It also illustrates the possibility that *Theileria* species may be masked by *T. parva* infections.

In a second experiment, two buffalo born and reared in captivity and four control cattle were inoculated with the *T. lawrencei* (Serengeti transformed) stabilate used in the first experiment. Three of the four cattle died of theileriosis and the fourth recovered spontaneously from severe theileriosis. The two buffalo had enlarged hyperplastic lymph nodes but no febrile response and, on microscopic examination of smears from the lymph nodes, no macroschizonts were detected. Attempts to isolate the parasite in cell culture and by tick feeding of the buffalo were successful. Few piroplasms were found in the erythrocytes of the buffalo. *Theileria*-infected cell lines were established from the control cattle. Using monoclonal antibodies raised against *T. parva* (Muguga) schizonts, six of six monoclonal antibodies reacted against the schizonts in the cell culture isolates from the cattle; whereas, only five of six monoclonals reacted with a series of cell culture isolates from both of the buffalo. This indicates that an antigenic in *T. lawrencei* can occur rapidly. These preliminary results show that *T. lawrencei* after adaption to cattle can still be infective to buffalo. The reactions of the buffalo appear to be similar to the reactions described by Brocklesby (1964) and Barnett and Brocklesby (1966) for the experimental infection of buffalo with *T. parva*. The development of a carrier state in these buffalo is being investigated.

Some of the adult buffalo from the Kabete breeding herd have been used for *T. lawrencei* isolation. *T. lawrencei* have been isolated over a period of 2 years from the same individuals. These isolates will be used for in vitro and in vivo studies on cross-immunity. In future, it is planned to use cloned *T. lawrencei* for the study of antigenic variation in buffalo and it is hoped that easier methods of identifiying antigenic types will be developed to replace expensive cross-immunity experiments in cattle.

Other *Theileria* Species Harboured by Buffalo

Buffalo harbour at least three species of Theileriidae, *T. lawrencei* (already discussed), *T. mutans*, and *T. velifera*. *T. velifera* has never been associated with disease and often occurs as a mixed infection with *T. mutans*. *T. mutans* has also been considered to be nonpathogenic to cattle. Some isolates, however, have been demonstrated to be pathogenic (Irvin et al. 1972; Kimber and Young 1977; Uilenberg et al. 1976). Paling et al. (1980) demonstrated that *T. mutans* isolated from buffalo caused a marked anemia in cattle. It is possible that *T. mutans* from buffalo may be more pathogenic to cattle than those maintained within cattle populations. Young and Moll (unpublished) found that in Masailand *T. mutans* was much more pathogenic to calves than had been previously believed. They monitored disease in calf populations from birth and found that a considerable proportion developed a severe anemia associated with infection, which in some cases was fatal and frequently affected the growth of the calves. There was strong evidence that *T. mutans* in this area could be derived from buffalo that have extensive contact with cattle. *T. mutans* infection in the buffalo tick *A. cohaerens* was found to be at a level of 15%. Further investigations into the pathogenicity for cattle of buffalo isolates of *T. mutans* need to be undertaken.

Eland and Theileriosis

Eland have been suspected of being involved in the epidemiology of cattle theileriosis for many years (Lichtenheld 1911; Beaumont 1939) because macroschizont and piroplasm indistinguishable from those of *T. parva* have been found in eland that apparently died of *Theileria* infections. Attempts to infect eland with *T. parva* have failed (Lawrence 1941; Lewis 1943; Brocklesby 1962). Attempts to transmit *Theileria* from eland to cattle have been made by Brocklesby (1962) and Irvin et al. (1972). Brocklesby did report the transmission of *Theileria* from eland to cattle using *Rhipicephalus pulchelus* on one occasion. On subsequent challenge with *T. parva* the cow proved to be immune. However, it is possible because of the nature of the infection that the cow acquired an accidental *T. parva* infection.

Irvin et al. (1972) attempted to transmit *Theileria* from infected eland to cattle. Although *Theileria* infection was demonstrated in the ticks (*R. appendiculatus* and *R. simus*), transmission to cattle was not observed. They concluded that under field conditions the *Theileria*-like parasites of eland were not transmissible to cattle.

A *Theileria*-like parasite described by Martin and Brocklesby (1960) and Brocklesby (1962) was associated with the death of an eland. The pathology of the eland was striking and the lesions were associated with the presence of the parasite. Martin and Brocklesby described the parasite as *Cytauxzoon*

taurotragi as the schizonts resembled those of *Cytauxzoon* first described by Neitz and Thomas (1948) in a duiker and subsequently in kudu (Neitz 1957) and giraffe (McCully et al. 1971).

Recent work has established that eland harboured a theilerial parasite infective to cattle (Young et al., 1977c). Macroschizonts and piroplasms occurred without any clinical signs other than enlargement of lymph nodes in the cattle receiving either *R. appendiculatus* or *R. pulchellus* adult ticks fed on parasitemic eland as nymphs. The sera from the cattle developed antibodies in the indirect fluorescent antibody test against antigens prepared from schizont-infected cell line isolated from eland but not against *T. parva* or *T. mutans*. The cattle recovered from the theilerial parasite from eland but died on challenge with a lethal dose of *T. parva*.

The same parasite was studied in experimental infection in eland born in captivity. In contrast to the situation in cattle, clinical signs of theileriosis developed in eland and of a total of 10 eland infected two died of theileriosis. In addition to these experimental cases, three naturally infected eland that died from theileriosis were studied. These findings were compared with the only previously described detailed case (Brocklesby 1962). Although the enormous macroschizonts or collection of cytomeres described by Brocklesby have not been observed in vivo in our studies, they have been observed in in vitro studies (Stagg et al. 1976). Therefore, we concluded that the parasite studied by Brocklesby is the same as the one described in our studies.

The *Cytauxzoon* syndrome may occur infrequently in eland. It was concluded that this species belonged to the genus *Theileria* and should be referred to as *T. taurotragi* (Martin and Brocklesby 1960). *T. taurotragi* has now been well characterized with further studies on its behaviour in eland (Grootenhuis et al. 1977, 1979, 1980), on its development in the tick (Young et al. 1980), and on its morphology and ultrastructure (Young et al. 1978).

Recently Grootenhuis, Young, and Martens (unpublished) under simulated natural conditions at Mombasa showed that *T. taurotragi* was transmitted from eland to cattle. *Theileria*-free cattle were introduced into a herd of eland and oryx. All cattle had mild *Theileria* infection and some had antibody response to *T. taurotragi* antigens.

Another finding of importance is the relationship of *T. taurotragi* with a collection of *Theileria* isolates from cattle that have yet to be shown to be associated with clinical theileriosis. These isolates have been *Theileria* sp. (Githunguri) (Burridge et al. 1974), *Theileria* sp. (Mwanza), and *Theileria* sp. (Idobogo) (Uilenberg et al. 1977). Antisera against all these parasites including *T. taurotragi* cross-react against antigens of *T. taurotragi* and *Theileria* sp. (Githunguri) and (Idobogo) in the IFA test (Young et al. 1977d, Grootenhuis et al. 1979). One of these parasites *Theileria* sp. (Idobogo) produced an infection in eland. It was concluded for biological and immunological reasons that it is likely that these parasites are strains of the same species (Grootenhuis et al. 1981).

These studies have defined to a certain extent the role of eland in the epidemiology of theileriosis. In conclusion it has been shown: (1) eland are not carriers of *T. parva* or *T. lawrencei*; (2) eland are carriers of *Theileria* species infective to cattle, *T. taurotragi*; the morphology of this parasite is similar to *T. parva* and can therefore cause confusion in the diagnosis of cattle theileriosis. *T. taurotragi* apparently does not cause disease in cattle nor does it protect cattle against fatal theileriosis (*T. parva* and *T. lawrencei* infection); (3) *T. taurotragi* of eland and *Theileria* spp. (Githunguri, Idobogo, and Mwanza) may represent strains of the same parasites adapted to different hosts. It would appear that *T. taurotragi* is widespread in Africa and probably has a wide (bovid) host range.

Other Wild Animals and Theileriosis

Cytauxzoonosis

Neitz and Thomas (1948) created the genus *Cytauxzoon*, as the description of *Theileria*-like parasites. Their observations were made on a duiker (*Sylvicapra grimmia*) that died from this parasitic disease. The parasite was defined as one that multiplies by schizogony in histiocytes and by division into four in erythrocytes.

Subsequently *C. strepsicerosi* was recorded in the kudu (*Tragelaphus strepsiceros*) (Neitz 1957), *C. taurotragi* in the eland (Martin and Brocklesby 1960), and *Cytauxzoon* in the giraffe (McCully et al. 1971). All these parasites were associated with death of the hosts from which they were described. Except in the case of *C. taurotragi*, no attempts were made to transmit these parasites to other animals, so nothing can be said of their host range or their importance in cattle theileriosis.

Theileria gorgonis of Wildebeest (*Connochaetes taurinus*)

This *Theileria* species was described by Brocklesby and Vidler (1961) from the blue wildebeest. Purnell et al. (1973) were not able to transmit this

parasite to cattle and it proved to be serologically distinct from *Theileria* species occurring in cattle (Burridge and Kimber 1973). It was also serologically distinct from *T. taurotragi* (Young et al. 1977d). This parasite was not associated with disease in wildebeest except after splenectomy and appears to be of no importance in the epidemiology of cattle theileriosis.

Theileria of Impala (*Aepyceros melampus*)

The occurrence of *Theileria* parasites in impala has been previously described by Neitz (1957), Brocklesby and Vidler (1966), and Irvin et al. (1973). Grootenhuis et al. (1975) did some experimental work and showed that *Theileria* from impala was not transmissible to intact or splenectomized cattle, but was readily transmitted between impala by blood inoculation. Serological comparison differentiated this *Theileria* from *T. parva*, *T. lawrencei*, *T. mutans*, *T. taurotragi* and *T. gorgonis*. This parasite was not associated with disease in impala and appears to be of no importance in the epidemiology of cattle theileriosis.

Other *Theileria* Species from Wildlife

Three other species have been named: *T. hippotragi* in roan antelope (*Hippotragus equinus*) by Todd and Wolbach (1912), *Tragelaphus stordyi* in Grant's gazelle (*Gazella granti*) (França, 1912), and *T. tragelaphi* in bushbuck (*Tragelaphus scriptus*) (Neitz 1931). *Theileria* piroplasms are very common in the Bovidae and it would be possible to create a large number of species based on different host records of intraerythrocytic piroplasms. However, this would contribute nothing to the knowledge of the epidemiology of theileriosis.

In Vitro Studies

Stagg, Dolan, Grootenhuis, Leitch, and Young (unpublished) have perfected a technique of in vitro infection of cattle cells with *T. parva* and *T. taurotragi* that results in the production of transformed cell lines infected with *Theileria* schizonts. In addition, they have used the same technique to attempt to infect eland, buffalo, waterbuck, bushbuck, wildebeest, sheep, and goat cells with the same parasites. In the case of *T. parva* only two types of cattle cells (*Bos indicus* and *Bos taurus*) and buffalo cells became infected with schizonts and whereas the cattle cells readily transformed, the infection in buffalo cells died out. In contrast, *T. taurotragi* infected the cells of all these species with the production of schizont-infected cells.

The results of the in vitro technique agree with the reported host range of *T. parva*. In the case of *T. taurotragi* it has indicated a wide host range that will have to be investigated in vivo.

The in vitro infection test could be a useful method to determine host range of known as well as newly isolated *Theileria* parasites. The cells become infected and transformed cells could be grown in bulk for detailed characterization of the parasite.

Discussion

In Kenya, one of the main goals of the Department of Veterinary Service is to improve control of cattle theileriosis. The work described in this review can contribute in two ways to this: (1) the development of a vaccination against theileriosis; and (2) a fuller understanding of the epidemiology of theileriosis.

The buffalo as a carrier of *T. lawrencei* could prove to be an important source of antigenic material for the immunization of cattle. In addition, it is not known how many antigenic types of *T. lawrencei* occur in buffalo, which could become adapted to cattle, and this impedes the development of effective vaccination against the buffalo parasite. It is likely that the buffalo will be the focal point in the research to produce effective vaccination against cattle theileriosis.

The majority of the 30 wild ruminants occurring in Kenya carry *Theileria* parasites. Some of these parasites are probably shared with domestic livestock as has been well demonstrated in the case of eland (Young et al. 1977d; Grootenhuis et al. 1979). Even in the case of domestic livestock, the *Theileria* parasites of sheep and goats have not been fully characterized. It is of importance for theileriosis control to identify these parasites and compare them with the *Theileria* parasite responsible for ECF. To undertake these studies properly, several new procedures and techniques need to be developed. The studies reported here illustrate the need for experimental wild animals for use in the characterization and isolation of *Theileria* species. The in vitro infection technique using panels of cells from a wide range of animals as developed by Stagg et al. (1976) may prove to be a practical tool for the study of the host range and for the characterization of these *Theileria* parasites. This technique could reduce the number of *Theileria*-free wild animals and cattle used in these studies. Also, the development of improved in vitro methods of characterization of *Theil-*

eria species and strains by immunological means are essential in this work. The use of monoclonal antibodies produced against *Theileria* schizont (Pinder and Hewett 1980) could be very useful for the antigenic characterization of *Theileria* parasites but requires considerable development.

As it is not possible or desirable to separate wild animals completely from domestic animals in Kenya or elsewhere in Africa, wild animal theileriosis must be considered in the control of domestic animal theileriosis or no effective methods of control will be developed. From the conservationist viewpoint it is necessary to determine which wild animals are to be implicated as dangerous disease reservoirs for livestock. In the past, wild animals have been considered to be the main source of certain diseases in livestock, but further studies have shown that wild animals are blameless. These facts are needed for establishment of land-use policies that are of equal importance to the farmer and the tourist industry.

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Potential Application of Research on African Trypanosomiases in Wildlife and Preliminary Studies on Animals Exposed to Tsetse Infected with *Trypanosoma congolense*

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Wildlife has an essential role to play in the future socioeconomic plans for developing countries. However, consideration must be given to the fact that some species of wildlife can act as reservoir hosts of pathogens of man and his domestic animals. Such dangers must be recognized and clearly defined.

Of all diseases, trypanosomiasis provides one of the most complex and emotive interactions between wildlife, man, and domestic livestock because some wild animals are known to act as reservoir hosts for the human pathogens *Trypanosoma rhodesiense* (Heisch et al. 1958) and *T. gambiense* (P. De Raadt personal communication), as well as for the important pathogens of domestic livestock, namely, *T. congolense*, *T. vivax*, and *T. brucei* (Ashcroft 1959). As a result, several trypanosomiasis control campaigns have involved large-scale destruction of wildlife. However, such indiscriminate slaughter has on each occasion failed to control the disease and in some cases has exacerbated the problem because tsetse have readily adapted to other hosts (Buyst 1977). It is obvious that such strategies have to be reconsidered and that much more precise data are required on the role of different species of tsetse, different species of trypanosomes, and different species of wildlife in the transmission of infection between wild animals, man, and domestic livestock in order that effective control measures can be enacted.

In the past, it was generally assumed that wildlife was refractory to most infections, but as surveillance of disease intensified, more evidence became available to show that this was not the case. For example, certain species are known to be highly susceptible to trypanosomiasis (Carmichael 1934; Ashcroft et al. 1959; Desowitz 1960; Godfrey and Killick-Kendrick 1967). Over the last century, wild animal populations have been dwindling rapidly in the face of human population pressure and, whereas, in the past large-scale losses through disease probably went unnoticed, at the present time, certain species are in danger of extinction. As a result, there is now a definite need to be able to diagnose, treat, or control diseases such as trypanosomiasis in individual animals. At the same time, the establishment of game ranching projects enhances the risk of disease, a situation that will increase requirements for accurate diagnosis and understanding of disease processes. Thus, to quote Baker (1968) "more information on the pathogenicity of the salivarian trypanosomes to game animals, and on the pathology of the infections produced, is badly needed."

Currently, it is felt that the exploitation of trypanotolerant livestock, i.e. animals with reduced susceptibility to trypanosomiasis, offers one of the most promising approaches to the control of animal African trypanosomiasis (FAO 1979; Tsetse and Trypanosomiasis Control 1980). It is now established that certain breeds of cattle, sheep, and goats exhibit this trait to differing extents (reviewed by Murray et al. 1979). Wild animals have a reputation for being even more resistant to trypanosome infection and, in some cases, it is believed that they might be completely refractory to infection. Such conclusions are usually based on both the fact that wildlife survives in areas heavily infested with tsetse and on surveys involving the collection of single blood samples examined for the presence of trypanosomes using a range of light microscope parasitological techniques. This approach yields little useful information on the question of susceptibility of different species to trypanosomiasis.

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Ideally, what are required are sequential studies carried out on animals, not previously exposed to trypanosomiasis, following experimental infection with properly characterized trypanosomes where clinical, parasitological, immunological, and pathological parameters are assessed. In one of the few studies of this type, Ashcroft et al. (1959) evaluated the susceptibility of various species of wild animals, in most cases not previously exposed, to fly challenge and, in a few cases, to needle challenge with *T. rhodesiense* and *T. brucei*. They found a wide range of susceptibility between species and also some variation within species. Some species, including Thomson's gazelle, dik-dik, blue forest duiker, jackal, bat-eared fox, antbear, hyrax, serval cat, and monkey, became infected and usually died. Another category included less susceptible animals or even species refractory to infection. These could be divided into species that became infected and had parasitemias of considerable duration such as the common duiker, eland, Bohor reedbuck, spotted hyena, oribi, bushbuck, and impala, species usually infectible but with scanty parasitemias such as warthog, bushpig, and porcupine, and the baboon, which was refractory.

A few attempts have been made to infect a small number of animals with *T. congolense* and *T. vivax*. With *T. congolense* some species such as oribi, bushpig, and porcupine were not infectible while others including the red-fronted gazelle, Bohor reedbuck, greater kudu, lesser kudu, waterbuck, Thomson's gazelle, steinbok, and elephant became infected and then recovered; a few animals such as jackals and certain species of monkeys were found to be highly susceptible and even died (Carmichael 1934; Ashcroft et al. 1959; Desowitz 1960; Roberts and Gray 1972). The number of species studied with *T. vivax* is even less. A single adult reedbuck did not become infected when challenged with *Glossina palpalis* infected with *T. vivax* (Desowitz 1960), while both duiker and red-fronted gazelle became infected but made a spontaneous recovery (Desowitz 1960; Roberts and Gray 1972).

Obviously much more data are required on the question of susceptibility of different species of wildlife to all the major African trypanosomes. Nevertheless, certain species show a remarkable degree of resistance to trypanosomiasis and offer an excellent model to study the mechanism(s) of host resistance with a view to possibly potentiating such traits in domestic livestock.

The availability of several species of wild animals reared in a tsetse-free environment by the Wildlife Diseases Section of the Veterinary Research Laboratories, Kabete, Kenya, has provided us with the unique opportunity to study many of the important questions raised in the preceding discussion. Accordingly, (1) Studies have been undertaken to evaluate the degree of susceptibility of different species of wild animals to *T. congolense*, *T. vivax*, *T. brucei*, and *T. rhodesiense*. This should provide knowledge on the impact of trypanosomiasis on wild animals per se as well as an evaluation of the possible role of different species as reservoir hosts for domestic livestock and for man. This study will include an investigation of infection rates and transmission characteristics of tsetse fed on trypanosome-infected wildlife. (2) Studies will be made of the mechanism(s) of susceptibility, including an evaluation of potential innate resistance factors as well as possible mechanisms of acquired resistance.

Reaction of Eland and Waterbuck to Bite of Tsetse Infected with *T. congolense*

In susceptible domestic animals and in man, a skin reaction develops at the site of challenge several days after the bite of a tsetse infected with trypanosomes (reviewed by Emery et al., in press). By analogy with the lesion considered pathognomonic of primary syphilis in man, which it resembles, this reaction has been termed the "chancre." From our studies in goats and cattle, it would appear that the skin acts as the site at which the parasites become established and proliferate prior to dissemination to the bloodstream. The intensity of the lesion is related to the number of trypanosomes inoculated into the skin and in our experience once a chancre has developed infection always follows. Thus, these early events are important in the establishment of infection and possibly affect the susceptibility of the host to the parasite. Consequently, a detailed examination of the lesion and associated changes has been undertaken in several wildlife species; as far as we are aware there is no published information on whether or not the chancre reaction develops in any species of wildlife apart from chimpanzees (Godfrey and Killick-Kendrick 1967).

In the present pilot study an eland and a waterbuck were bitten by *G. morsitans morsitans* infected with *T. congolense*. The eland and waterbuck were females aged 6 months and 14 months, respectively; both had been reared at Kabete and had no previous experience of trypanosomiasis.

Five infected flies were allowed to feed on the shaved flanks of each animal and subsequently these ten infected flies were fed on the flanks of two susceptible East African goats, five on each animal. Five uninfected flies were fed on the contralateral flank of each animal. The bite of the uninfected tsetse produced no detectable reaction in any animal,

whereas, with infected flies skin reactions developed at 9 of the 10 bite sites on goats, but only two of the five bite sites on the eland and three of the five bite sites on the waterbuck.

The kinetics of development of these early changes as they occurred in the eland and waterbuck (Fig. 1) were quite different from the pattern observed in the goat and in the bovine, the two domestic animal species we have studied most extensively (Emery et al., in press).

In the goat, the local skin reaction that developed from the bite of a tsetse infected with *T. congolense* was first detectable as a palpable discrete nodule 6–7 days after challenge. The lesion progressively enlarged and attained a more diffuse raised plaque with substantial subcutaneous oedema, heat, and pain, being most marked by about day 10. Thereafter, the reaction subsided rapidly and 5 days later

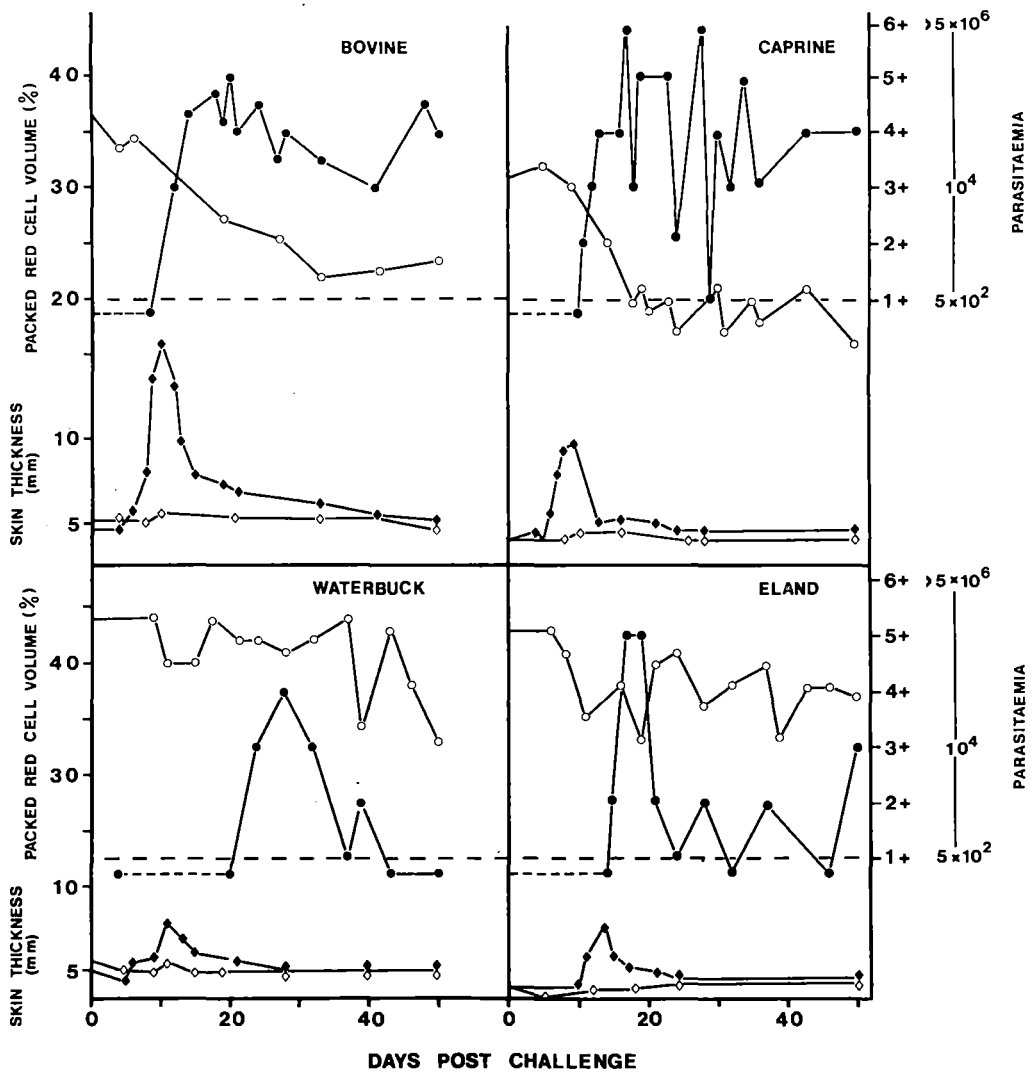

Fig. 1. Kinetics of development of the chancre in a bovine, a goat, a waterbuck, and an eland following challenge with G. m. morsitans infected with T. congolense: ◆ skin thickness at site of bite by infected tsetse; ◇ skin thickness at site of bite by uninfected tsetse on contralateral flank; ○ packed red cell volume (%); • parasitemia as detected by the method devised by Murray et al. (1977) using the scoring system described by Paris et al. (1980). The broken line indicates the limit of sensitivity for detection of trypanosomes.

the skin was almost back to normal. The skin had increased in thickness from 4.0 mm to reach 9.8 mm at the peak of response on day 10 (Fig. 1). The skin reaction that occurred in susceptible Boran, Friesian, and Charolais following the bite of a *T. congolense*-infected tsetse was similar to that described for goats. In some cases, the lesion reached as much as 100 mm in diameter at its maximum, by which time, skin thickness had more than doubled (Fig. 1).

In the eland, the skin reaction was mild and was detectable only by an increase in skin thickness without being indurated or circumscribed. The increase in skin thickness became apparent only on day 11 after the tsetse bite, reaching peak thickness on day 14 (from 4 mm to 7.8 mm) and then returning to normal by day 24 (Fig. 1).

In the waterbuck, a skin reaction was first detected on day 7 after tsetse challenge. The reaction was clearly observable on day 9 and by day 11 it attained its peak size of about 30 mm in diameter, with skin thickness increasing from 5 mm to 8.6 mm (Fig. 1). Thereafter, the lesion subsided and had disappeared by day 20.

The initial detection of the skin reaction always preceded the appearance of parasitemia. In the goat, the prepatent period ranged from 9 to 11 days, in cattle from 13 to 15 days, in the eland 13 days, whereas in the waterbuck parasites were not detected in the blood by microscopical examination until day 24.

In all four species, the appearance and development of the chancre was accompanied by significant enlargement of the draining prefemoral lymph node; the response, however, was more pronounced in goats and cattle.

In summary, the development of skin reactions following the bite of tsetse infected with *T. congolense* has been recognized for the first time in wildlife. A significant difference in the early events was found between domestic livestock and wild animals. There was some evidence that the transmission rate of the parasite was lower, i.e. the inoculation of trypanosomes from the tsetse into the skin of the host was not as successful in wild animals — only about half of the successful tsetse feeds in the eland and waterbuck resulted in the production of a skin reaction, a response considered indicative of the establishment of infection (Emery et al., in press). In domestic animals most tsetse feeds resulted in the production of chancres.

Furthermore, the size and severity of the skin reaction was significantly less in the wild animals. It is possible that this was related to the number of trypanosomes that became established in the skin as it has been shown, using intradermal needle inoculation of different numbers of trypanosomes, that the size and intensity of the reaction is dose dependent (Emery et al., in press). Alternatively, it might be that the rate of replication of the parasite was slower in the skin of these wild animals. In the waterbuck, despite the fact that trypanosomes were detected in the skin as early as day 11[3] they were not seen in the blood until day 24, further confirming the importance of the skin in the restriction of the trypanosome prior to bloodstream dissemination and indicating a possible role for skin reactivity in host susceptibility to trypanosomiasis.

The possible ways in which the skin might achieve this are not known but differences in skin structure do exist between different breeds of cattle and these may influence susceptibility. Carr et al. (1974) found an association between skin thickness and the prevalence of trypanosomes; within herds of East African Zebu, *T. congolense* infections were more common in thinner skinned animals. It is also known that skin vasculature can vary between certain indigenous African breeds of cattle and imported breeds (Amakiri 1976), the former usually being less susceptible to trypanosomiasis. We feel that the role of the skin in the transmission and establishment of infection is significant and that further studies on wildlife might elucidate the factors that influence these processes.

Another significant feature to emerge from this study was that the level and duration of parasitemias were less in the wild animals (Fig. 1). The level of the first peak of parasitemia in the eland was 10^5 trypanosomes per millilitre and in the waterbuck 5×10^4 trypanosomes per millilitre; whereas, in goats and cattle the level was as high as 10^6 trypanosomes per millilitre; the levels of the subsequent peaks of parasitemia were also less in the wild animals. The duration of sustained parasitemia in the eland and waterbuck was shorter as judged by the fact that they became negative for trypanosomes earlier than domestic animals and were negative on many more occasions over the period of 90 days up to the time this article went to press.

Associated with the appearance of parasitemia in goats and cattle, a significant drop in packed red cell volume (PCV) developed and this was progressive (Fig. 1). The majority of these animals had to be treated to prevent death. On the other hand, in both the eland and waterbuck, while a small drop in PCV occurred in association with parasitemia, no significant anemia developed and the PCV levels were soon back within normal range. An alternative explanation for the transient drop in PCV was that it was not related to parasitemia but was the result of repeated tranquilization and venipuncture.

[3]This was demonstrated by subinoculation into mice of a suspension prepared from a biopsy of a day 11 reaction.

There is evidence from work in cattle and in mice that the capacity to control parasitemia might be related to a superior immune response to the trypanosome. Studies in N'Dama (a trypanotolerant breed of cattle), have indicated that these cattle have the ability to acquire and mount a greater immune response than Zebu (Desowitz 1959). At the same time, work on mice indicates that reduced susceptibility and the greater ability to control trypanosome parasitemia is associated with superior immune responsiveness and the ability to produce IgM antibodies (Murray et al., in press). Furthermore, studies at ILRAD on sera from N'Dama and Zebu suggest that the recognition of a small number of common trypanosome antigens by the animal's humoral immune system is correlated with N'Dama's greater capacity to control and abort trypanosome infections (Shapiro and Murray, in preparation).

In the current project, we intend to evaluate the immune response to the trypanosome of wildlife hosts of different species and also to investigate if wild animals that recover from trypanosome infections recognize the same common antigens as trypanotolerant cattle. Such antigens might be of major importance in developing new immunodiagnostic tests or even vaccines.

It should also be considered that many species of wild animals could be poor hosts for trypanosomes for non-immunological reasons such as the absence of essential nutrients or the presence of deleterious factors in their bloodstreams. Some of these factors have been recognized already. It has been shown that cotton rats are completely refractory to *T. vivax* because of a trypanocidal factor identified as a serum macroglobulin (Hudson and Terry 1970). The only way in which *T. brucei* can be distinguished from *T. rhodesiense* is by its inability to infect man; the lytic host factor responsible has been identified as a high density lipoprotein (Rifkin 1978). Other factors that could be important in host susceptibility to trypanosomiasis might include complement reactivity and the activity of the mononuclear phagocytic system. The fact that certain species of wildlife are resistant or even refractory to trypanosome infection offers an opportunity to investigate the existence of such factors. Their identification and isolation might be important for future use in the management of the disease in man or in domestic livestock.

It is likely that there are several other factors that influence host survival in endemic tsetse areas and thus contribute to the overall picture of trypanotolerance. It is well established that certain species of tsetse exhibit definite host feeding preferences (Weitz 1963), although these traits are by no means stable and can be influenced by environmental factors (Moloo 1973; Moloo et al. 1980). The significance of such tsetse preferences was shown recently by Roberts et al. (1980) when they compared the attractiveness of cattle and oryx to tsetse under critical experimental conditions. They found that five times as many flies were attracted to cattle and they were able to count full engorgement on cattle of 279 tsetse during the period of observation; only 4 tsetse were seen to obtain partial blood meals from the oryx during the same time.

Other factors important for survival are likely to include the capacity to forage and utilize food and the ability to regulate body temperature and conserve water, traits well developed in wild animals (EAVRO 1967). Recognition and understanding of all these traits must yield essential information that will aid future control of trypanosomiasis.

In conclusion, it appears to us that nature has presented to man a vast animal kingdom whose excitement and beauty can be viewed by all. She has achieved this by a rigorous process of selection for important characteristics that allow survival under the most severe ecological pressures. The recognition and delineation of the mechanisms underlying these characteristics will not only help us in our battle to conserve the wild animal kingdom but also aid us in developing new tools to treat, control, and even prevent some of the most important diseases that afflict man and his domestic animals.

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The Role of Wild Ruminants in the Epidemiology of Nematodiasis in Kenya

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It has been known for many years that, on the basis of taxonomic criteria, wild ruminants and domestic ruminants share a proportion of their nematode fauna. However, studies on cross infection between the two groups have been performed experimentally on only a limited scale and even less is known about the extent to which this occurs naturally in the field.

Inevitably, therefore, any assessment of the role of wild ruminants in nematodiasis must at this stage be based largely on knowledge of the disease in domestic animals and certain hypotheses regarding the expected degree and influence of cross infection.

On the basis of taxonomic criteria it has been estimated that about 20–40% (Sachs et al. 1973; Prestwood et al. 1976; Woodford 1976) of the nematode species commonly recorded in wild ruminants are also found in domestic animals. However, of these species those likely to be of most economic significance are: (1) gastrointestinal — *Haemonchus* spp.; *Trichostrongylus* spp.; *Impalaia* spp.; *Cooperia* spp.; *Nematodirus* spp.; *Oesophagostomum* spp.; and *Trichuris* spp.; and (2) lungworms — *Dictyocaulus* spp.; *Protostrongylus* spp.; and *Muellerius* spp.

The first demonstration of experimental cross infection was in 1926 by Le Roux who infected two lambs with the larvae cultured from the feces of a Roan antelope and a blue wildebeest and later from the feces of an impala (Le Roux 1930). Subsequently a series of experiments in the 1930s by Monnig demonstrated that a number of species, including *Haemonchus*, *Trichostrongylus*, *Nematodirus*, and *Cooperia* species, could be readily transmitted experimentally to sheep (Monnig 1931, 1932, 1933).

In 1979, Horak successfully infected sheep with the larvae of *Haemonchus*, *Trichostrongylus*, and *Impalaia* species cultured from the feces of naturally infected blesbok. Similarly, sheep, goats, and calves were infected with the larvae of *Haemonchus*, *Longistrongylus*, *Trichostrongylus*, *Impalaia*, and *Cooperia* species cultured from impala feces. Only the sheep and goats could be successfully infected with the *Cooperioides* and *Oesophagostomum* of impala origin.

In two experiments involving 15 Thomson's gazelles and 16 Merino sheep (Preston et al. 1979) it was shown that of the 11 species that established in gazelles from the larval culture of gazelle feces only three species, i.e. *Haemonchus*, *Trichostrongylus*, and *Cooperia*, were infective for sheep. From the sheep fecal culture only *Haemonchus* became established in both gazelles and sheep, but the results of fecal egg counts (Fig. 1) and measurements on weight, length, and spicule lengths (see Tables 1 and 2) indicated that either the gazelles had developed a better innate or acquired immunity or that

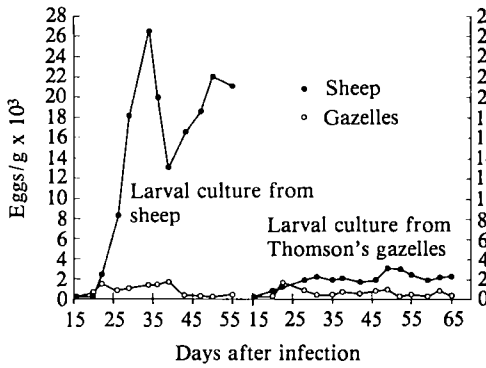

Fig. 1. The fecal egg counts of Merino sheep and Thomson's gazelles following infection with larvae cultures from sheep and gazelle feces at an infection rate of 200 larvae per kilogram of body weight: • Merino sheep; ○ Thomson's gazelle.

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H. contortus was primarily a sheep parasite that became established to a much lesser extent in the gazelle.

Table 1. The results of mean worm recoveries in Merino sheep and Thomson's gazelles infected with larvae cultured from naturally infected sheep and gazelles.

Host	Sheep culture	Gazelle culture
Sheep	24%	4% (3.9% *H. contortus*)
Gazelles	3%	14% (1.5% *H. contortus*)

Table 2. The mean weights, lengths, and spicule measurements of *H. contortus* worms recovered from Merino sheep and Thomson's gazelles following experimental infection with larvae cultured from both sheep and gazelle feces.

	Larvae cultured from sheep		Larvae cultured from gazelles	
	Sheep	Gazelles	Sheep	Gazelles
Weight (mg)	1.02	0.18	0.93	0.16
Length (mm)	19.21	11.15	18.31	12.28
Spicules (μm)	461	422	456	435

Economic Significance and Future Role

To assess the economic significance for domestic animals of nematodiasis in wild ruminants it is necessary to know what extent the wild ruminants contribute to overall pasture larval contamination and what additional effect this produces on the domestic stock. Because this information is not yet available, it is only possible to make estimates using the existing knowledge and to make hypotheses for the future.

Using the data known about Merino sheep (Allonby and Urquhart 1975) and what is known about Thomson's gazelles (Preston et al. 1979) it can be estimated that in an area grazed by 900 sheep and 100 gazelles that the gazelles' share of the strongyle egg output on the pasture would not exceed 0.5%. From this it must be concluded that under existing methods of control the gazelles do not play a significant part in the epidemiology of sheep helminthiasis.

However, the gazelles do act as a reservoir host so that if frequent anthelmintic treatment were adopted in an attempt to eradicate the disease, this could not be achieved because of the presence of the gazelles. Although this may not be of practical significance at present, there is evidence from recent experiments (Allonby and Preston, unpublished) that such a regimen, i.e. of producing progressively "helminth-free" pasture, may be economically feasible and could provide scope for the use of an irradiated larval vaccine as a means of immunological control (Jarrett et al. 1960; Mulligan et al. 1961). Under such circumstances, the presence or absence of wild ruminants acting as reservoir hosts would determine the success of this approach.

Furthermore, as it is now being recognized that the subclinical effects of nematodiasis are much greater than had been previously thought, the role of wild ruminants as reservoir hosts is likely to assume more importance and greater consideration will have to be given to their role in this important disease.

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Helminths in Wild Ruminants in Central Africa: Impact on Domestic Ruminants[1]

M. Graber[2]

Central Africa covers an immense area that includes Chad, the Central African Republic, and Northern Cameroon. It is divided into four climatic zones:

(1) A desert zone, north of the 15th parallel, covering, in Chad, the Borku, Ennedi, and Tibesti regions. With the exception of Borku, these are mountainous areas where rainfall is rare and irregular. Vegetation is sparse and in general there are few animals.

(2) A middle Sahelian zone called the steppe zone, from the 11th to the 15th parallel. Covered in the north with barren, sandy dunes, in the south it forms a vast, flat plain, rising in the west (Cameroon) and in the east (Guerrah, Ouaddai). The climate is tropical, with a single rainy season lasting from July to October. Annual rainfall ranges between 250 and 750 mm, and the region is rich in pasture lands and becomes increasingly wooded in the south. Here and there, the streams are lined with arcade-like stretches of relatively dense forest growth.

(3) A Sudanese savanna zone, from the 7th to the 11th parallel, straddling Chad and the northern regions of the Central African Republic. It is a flat land, dotted with a few hills and plateaus. In the south there is a large mountainous massif comprising the Yade Mountains in the west and the Bongo Mountains in the east. The rainy season lasts 4 months (end May to end September) and rainfall ranges from 900 to 1300 mm. The vegetation is of the savanna type; areas with scant tree and shrub growth are interspersed with wide bands of grassy pasture land.

(4) A Guinean zone, from the 3rd to the 7th parallel. The rainy season lasts 6 months and rainfall ranges from 1300 to 1600 mm. Generally, it is a wooded savanna that turns into dense forest stretches along the rivers.

The granitic barrier of the Yade and Bongo Mountains in the Central African Republic marks the dividing line between the streams flowing northward to Lake Chad through the Chari and its tributaries (Lim, Pende, Bahr Sara, Bahr Aouk, Bahr Salamat), and southward to Zaire through the Ubangui, Sangha, and Lobaye.

The best area for herding domestic ruminants lies between the 9th and 15th parallels, in Chad and Northern Cameroon. In Chad, prior to the great drought of 1972–74, there were, between the 13th and 16th parallel, some 4 million zebus, 4 million sheep and goats, and 350 000 camels. Most of these animals belonged to nomadic herders who travelled northward in the rainy season, and southward in the dry season, in search of richer pastures and water. These same areas are frequented by many wild ruminants (25 different species) that often wander along the same trails.

In the Central African Republic, domestic herding is a fairly recent activity. The first Bororo herdsmen settled in the Yade savanna in 1924. Since then, they have spread throughout the western half of the country. The number of bovines is estimated at 800 000. In the eastern half, a large number of wild animals of many different species occupy a vast hunting zone covering 320 000 km^2. This area is fairly isolated, sparsely populated, and herding is nonexistent. However, the northern section (Bahr Aouk) is at times invaded by clandestine herds from Chad and the Sudan. It is also crossed by two cattle import routes: one that goes from Birao to Bangassou via Ouadda and Bria; and, farther to the west, one that goes from Sahr in Chad to Bangui via Ndele, Fort-Crampel, and Fort-Sibut.

The fact that both domestic and wild ruminants are found in the same areas and along the same trails poses a number of problems, and we considered it worthwhile to determine what impact, if any,

[1]This paper was originally in French; with the author's permission, it was translated into English by IDRC.

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Table 1. Number of autopsies performed on domestic ruminants.

	Zebu					
	Suckling calves	Young bulls	Adults	Sheep	Goats	Camels
Chad	3955	1690	4743	5382	781	159
Central African Republic	422	123	627	38	4	—
Northern Cameroon	106	—	409	—	—	—

internal parasites infecting wild herbivores were having on domestic ruminant herds.

Tables 1 and 2 present figures for autopsies performed in Central Africa from 1954 to 1972. The helminths that were found were examined in accordance with standard methods: clearing with lactophenol for nematodes, staining with carminic acid, and serial sectioning for cestodes and trematodes.

It was found that a large number of helminths in wild ruminants are specific and affect domestic ruminants very rarely or not at all. These include:

(1) Nematodes: almost all the *Setaria* of the peritoneum, with the exception of *Setaria labiatopapillosa*, observed only twice in the buffalo in Chad and the Central African Republic (a single specimen in each case); the *Elaeophora* of the circulatory system; *Ashworthius lerouxi*, *Parabronema skrjabini*, *Ostertagia thalae*, *Haemonchus vegliai*, and all the *Longistrongylus* of the fourth stomach; *Bunostomum dentatum*, *Agriostomum cursoni*, *Paracooperia daubneyi*, and *Cooperia fuelleborni* of the small intestine; several esophagostomes (*Oesophagostomum synceri* and *Oesophagostomum lechwei*) of the large intestine and *Pygarginema africana*; *Pneumostrongylus cornigerus* of the respiratory system.

(2) Cestodes of the small intestine (*Moniezia monardi*, *Avitellina edifontaneus*, *Avitellina sandgroundi*, *Avitellina buechneri*) and bile ducts (*Crossotaenia baeri*).

(3) Trematodes of the first stomach: *Cotylophoron macrosphinctris*, *Carmyerius endopapillatus*, *Carmyerius schoutedeni*, *Carmyerius exoporus*, *Carmyerius minutus*, and *Stephanopharynx coilos*. *Schistosoma margrebowiei* parasitizes only the kobs (Buffon's kob and waterbuck) and it has been recorded only in southern Chad, in the Kyabé area between the two tributaries of the Chari, the Bahr Salamat and the Bahr Aouk.

Conversely, several zebu and sheep parasites have never been found in Central African wild ruminants, in particular *Schistosoma mattheei* and *Onchocerca gutturosa*, as well as some very abundant, very pathogenic nematodes: *Bunostomum phlebotomum* and *Oesophagostomum radiatum* found in the zebu and *Bunostomum trigonocephalum* found in sheep (Table 3). As far as sucking calves are concerned, there are two parasites that

Table 2. Number of autopsies performed on wild ruminants.

	Central African Republic	Chad	Northern Cameroon
Babalus (Syncerus) caffer	74	13	1
Alcelaphus lelwel	14	20	1
Damaliscus korrigum	1	8	—
Sylvicapra grimmia	3	2	2
Ourebia ourebi	5	4	4
Redunca redunca nigeriensis	2	8	—
Adenota cob	5	15	2
Kobus defassa	7	18	—
Gazella dorcas	—	37	—
Gazella rufifrons	—	21	—
Gazella dama	—	9	—
Hippotragus equinus	7	13	1
Oryx algazel	—	9	—
Addax nasomaculatus	—	1	—
Taurotragus derbianus	3	—	1
Tragelaphus scriptus	4	—	1
Strepsiceros strepsiceros	—	2	—

Table 3. Rate of infestation (%) of *Bunostomum phlebotomum*, *B. trigonocephalum*, and *Oesophagostomum radiatum*.

	Chad	Central African Republic	Northern Cameroon
B. phlebotomum			
Young bulls	55	56	—
Adults	5	4	7
B. trigonocephalum	1	52	—
O. radiatum			
Young bulls	51	53	—
Adults	15	38	10

Table 4. Rate of infestation (%) of *Toxocara vitulorum* and *Strongyloides papillosus* in zebus.

	Chad	Central African Republic	Northern Cameroon
T. vitulorum	0-20[a]	33	32
S. papillosus[b]	0-8	11	2

[a]Depending on region.
[b]In camels 27%; sheep 12%.

cause serious losses every year (5-20%): *Toxocara vitulorum* and *Strongyloides papillosus* (Table 4).

Autopsies on wild ruminants are normally performed on adult animals, less often on young ones, and never on sucking calves. Information on the latter age group is therefore completely lacking, and it is desirable that in future at least some fecal examinations be performed, especially among very young buffalo, because it is not difficult to find the eggs of *Toxocara vitulorum*.

Some other parasites are also common to both wild and domestic ruminants.

Dicrocoelium hospes is found in buffalo (16%), sheep (0.2%), and zebus (14-15%). It is found throughout the Central African Republic, in southern Chad, and in northern Cameroon below the 10th parallel. The role of the buffalo in the epidemiology of bovine and ovine dicrocoliasis appears limited. These animals are rarely found in the most affected areas, where domestic herding is fully or semi-sedentary (northern Cameroon, southwest Chad, western Central African Republic). Elsewhere (southeast Chad, northeast Central African Republic), the first intermediate hosts, the Achatinae of the genus *Limicolaria* resume their activity only in the wet season, at a time when the transhumant herds have already returned to the north. This explains why the disease is nonexistent in this type of herding. In these areas, it is likely that dicrocoliasis is maintained from buffalo to buffalo to the exclusion of any other domestic or wild ruminant.

The trematode *Fasciola gigantica* is also common to both wild and domestic ruminants (Table 5). It is much more widespread in wild ruminants where the disease strikes mostly waterbuck (40%), buffalo (38%), hartebeest (6%), Buffon's kob and *Hippotragus* (5%).

In the domestic herding zone, dissemination of the parasites is effected by both the domestic and wild Artiodactyla which, during the dry season, use the same trails. But this is not always the case, and in the southern part of the Central African Republic hunting zone, some centres of infection are far removed from cattle trails, and it may be supposed that propagation of hepatic distomatosis is effected by wild ruminants, especially the buffalo.

Schistosoma bovis, nonexistent in the Central African Republic, is frequently encountered in Chad in the camel (5%), sheep (11%), and zebu (34%), and in northern Cameroon in the zebu (35%). In these same areas, the percentage of wild herbivores that are infested is only 2.8% (Buffon's kob, waterbuck, buffalo, *Hippotragus*, *Damaliscus*, reedbuck). This indicates that the role of wild ruminants in the epidemiology of this infestation is not very important. As Dinnik pointed out in 1965, this trematode is more likely to infest domestic ruminants.

Table 5. Rate of infection (%) of *Fasciola gigantica*.

	Chad	Central African Republic	Northern Cameroon
Zebu			
Young bulls	3	28	—
Adults	28	62	43
Sheep	0.8[a]	2	—

[a]These low percentages are due to the fact that infested sheep often die of acute distomatosis and therefore never reach the slaughter house.

This is not true of the nonspecific Paramphistomidae and Gastrothylacidae (Table 6). Except in northern Cameroon, Gastrothylacidae appear to be associated with wild ruminants, and all the species examined, except for the crowned duiker, are hosts to a few. The rate and level of infestation of domestic herbivores is much lower, especially in the Central African Republic and in the northern section of Chad's Sahelian zone.

Like *Fasciola gigantica*, the Paramphistomidae gastric trematodes are very widespread among both wild and domestic herbivores. The intermediate host is *Bulimus*, which often coresides with the *Limnaea* that transmit hepatic distomatosis. In Central Africa,

Table 6. Percentage of animals parasitized.

	Paramphistomidae	Gastrothylacidae
Chad/Northern Cameroon		
Zebu	13–48	2[a]–29[b]
Sheep	22	1.2
Wild ruminants	35	15
Central African Republic	33–86	4
Zebu	13	—
Sheep	44	43
Wild ruminants		

[a]Chad.
[b]Northern Cameroon.

the reproduction rhythm of *Bulimus* is similar to that of *Limnaea*. In Sahelian and Sudanese zones, the multiplication period begins in September–October and spans almost all of the dry season, ending in May or June. Paramphistomosis and distomatosis are dry-season diseases contracted by a large variety of animals (including a great many wild ruminants) while they graze marshy regions, the bottoms of drying lakes and ponds, and the shores of certain rivers.

The average rate of hepatic stilesiosis in wild ruminants that are hosts to *Stilesia hepatica* is 12%. In the domestic herding areas of Chad, it definitely exceeds the rate in domestic animals, which is 0.06% in bovines, 2.4% in ovines, and 0.5% in goats. This indicates that, at least in these regions, hepatic stilesiosis is ascribable to wild rather than domestic ruminants. The latter are contaminated in areas where antelopes are numerous, especially near streams and lakes, i.e., in humid areas (Chari and tributaries). As early as 1911, Gough wrote in this regard: "it is probable that *Stilesia hepatica* was originally a parasite of the wild ruminants, and we may suppose that it adapted secondarily to sheep. The fact that this parasite is not reported in other parts of the world, its presence in antelopes, its numerous hosts, lead us to believe that in the beginning it was not a parasite of sheep."

Intestinal taeniasis in wild herbivores affects on the average one in five animals. It is more consequent in Chad (< 1%) than in the CAR (16%). Its nature is not the same: in the Central African Republic, *Stilesia globipunctata* and *Moniezia* are much less in evidence (less than 1.5%) than *Avitellina* (15%). In Chad and northern Cameroon, *Avitellina* and *Stilesia globipunctata* are found in equal numbers (9%), with *Moniezia* and *Thysaniezia* being much less numerous (1–2.5%).

The importance and the structure of intestinal taeniasis in domestic ruminants are completely different. In western Central African Republic, in the zebu, the disease is due to *Moniezia* and *Thysaniezia* cestodes, the average rate of infestation is 3%. In Chad and northern Cameroon, about 15% of zebus are parasitized, with *Thysaniezia* and *Moniezia* again predominant, as they are in goats. In sheep, 67% of which are affected, the species most commonly encountered are *Avitellina centripunctata* (43%), *Stilesia globipunctata* (35%), and *Moniezia expansa* (16%). In camels, 58% of which are affected, *Stilesia* are most numerous (28%), followed by *Moniezia* (15%), *Avitellina* (11%), and *Thysaniezia* (2.5%). In domestic herding, monieziosis and duodenal stilesiosis are particularly serious in sheep and camels. In areas inhabited by camels, the antelopes always harbour a large number of cestodes whose eggs they disseminate as they move toward their dry season pastures in the northern section of the Sahelian zone.

Cysticercosis due to *Cysticercus tenuicollis* (adult form: *Taenia hydatigena* of the dog) is also, in northern Chad, a common disease in sheep, goats, gazelles and, to a lesser degree, camels. In central and southern Central Africa, cysticerci collected from many antelopes have the larval shape of the *Taenia* of the lion, *Taenia regis*.

In Chad, systematic research has been carried out on nonspecific nematodes, which include:

(1) *Gaigeria pachyscelis*, a common parasite in sheep and goats (6–8%), found in the intestine of three buffalo (a few specimens).

(2) *Skrjabinema ovis*, a very rare oxyuridae in the sheep, goat, and hartebeest.

(3) *Impalaia tuberculata* of the zebu (0.1%) and camel (14%) and *Haemonchus longistipes* of the camel (72%), found in four gazelles in the north.

(4) *Cooperia punctata* and *Cooperia pectinata*, trichostrongyles of the zebu (10–20%), found in two hartebeest and one waterbuck.

(5) *Trichuris globulosa* and *Oesophagostomum [Proteracrum] columbianum* (Table 7) are well known in the Sahelian zone wherever there is herding of sheep and camels. Like the large intestine cestodes, *Impalaia tuberculata* and *Haemonchus longistipes*, they also affect the antelopes that have adapted to these dry areas. They are much rarer in central and southern Chad.

Table 7. Percentage of infestation of *Trichuris globulosa* and *Oesophagostomum columbianum*.

	T. globulosa	*O. columbianum*
Camel	34	28
Zebu	1–7.5	—
Sheep	4.5	40
Goat	3	15
Antelope	8	7.5

(6) *Haemonchus contortus*, a parasite of the fourth stomach, deserves particular attention. This is a Trichostrongylidae, with a short evolution cycle, that is well established in the Sahelian zone where it affects 20-50% of zebus, 37% of sheep, 35% of goats, 0.7% of camels, and 20% of antelopes, principally gazelles and oryxes. Many specimens have also been collected from hartebeest in Sudan and waterbuck in southern Chad near the border with the Central African Republic. *Haemonchus contortus* is therefore numerous and widespread in both domestic and wild herbivores.

Larval sparganosis is unknown in domestic ruminants. For wild ruminants, no in-depth studies have been made. It is very likely that it exists in Central Africa because the corresponding adult cestode, *Diphyllobothrium theileri*, has been identified on several occasions (lion in the Central African Republic; cheetah, jackal, and panther in Chad).

Larval echinococcosis due to *Echinococcus polymorphus* (adult, *Echinococcus granulosus granulosus* of the dog and jackal) is very rare in Central African wild ruminants. In Chad, the only known case is that of an oryx sacrificed in northern Ouaddai in 1965. In domestic ruminants, for the area as a whole, rates of infestation are low (zebu 1.4%; sheep 0.05%; goat 0%) except in the camel (57%). In the Central African Republic, no case of larval hydatidosis has been observed in either domestic or wild ruminants. However, in the northern section of the hunting zone, warthogs have been affected. The corresponding adult cestode could be a particular strain of *Echinococcus granulosus granulosus* adapted to the lion, with the possibility of a special lion/warthog cycle.

Mammomonogamus nasicola, a gapeworm of the larynx and pharynx, was found to affect 25% of the animals autopsied at Bouar (Central African Republic). The same parasite was found in a single buffalo in the eastern part of the Central African Republic. Information is still too fragmentary to allow definite conclusions to be drawn.

Generalized cenurosis due to *Coenurus cerebralis* (adult, *Taenia multiceps* of the dog) is the exception in Central Africa; it has been found only in the sheep and goat in Chad (0.1%).

Muscular cysticercosis involves two different parasites:

(1) *Cysticercus bovis*, an inermous cysticercus whose corresponding adult, *Taenia saginata*, resides in man's intestine. This zoonosis affects mainly the zebu: Chad, 8.5-19.5%, depending on the age group; northern Cameroon, 18.9%; and the Central African Republic, 21-49%. It is rare in wild ruminants: in Chad, only three observations have been recorded, in two gazelles in northern Ouaddai and in a Buffon's kob from the N'Djamena (Fort-Lamy) area.

(2) On the other hand, Central African wild ruminants are often carriers of armed cysticerci whose adult cestodes, *Taenia hyaenae* and *Taenia crocutae*, parasitize hyenas and more rarely *Lycaon*. The average rate of infestation is 15% in the Central African Republic and 8.4% in Chad and northern Cameroon. All species, except some gazelles, are affected. In domestic ruminants (zebu and camel), the number of animals affected is only 0.7%.

This type of cysticercosis is therefore most common in wild animals and hardly affects domestic Bovidae.

Conclusions

In Central Africa (northern Cameroon, Chad, Central African Republic), internal parasitism of wild herbivores is in most cases due to specific helminths that affect domestic ruminants only rarely or not at all. Conversely, certain pathogenic parasites frequently found in zebus and sheep do not exist among wild ruminants.

Where zoonoses are concerned, the role of wild herbivores is particularly discrete.

Nevertheless, certain infestations occur in both groups of herbivores. They are: hepatic distomatosis due to *Fasciola gigantica*; gastric paramphistomatosis due to Paramphistomidae and Gastrothylacidae; hepatic stilesiosis due to *Stilesia hepatica*; in northern areas, duodenal stilesiosis due to *Stilesia globipunctata* and monieziosis due to *Moniezia expansa*; haemonchosis of the fourth stomach due to *Haemonchus contortus* and to a lesser degree, trichuriosis due to *Trichuris globulosa* and nodular esophagostomiasis due to *Oesophagostomum columbianum*.

In areas where domestic and wild herds come into close contact, these diseases are liable to have certain repercussions on domestic herding. Here wild ruminants serve as parasite reservoirs and are liable to disseminate and disperse a number of parasites over large areas.

This mode of contamination mainly affects herds belonging to nomads because, with sedentary herds, the constant presence of humans means that fewer wild herbivores are normally present. In drawing up any plans for prevention, this situation must be kept in mind. Treatments must be adapted to the nature of the parasite transmitted by the wild ruminants. This treatment may be provided through the use of wide-spectrum drugs capable of destroying simultaneously hepatic and gastric distomes, intestine and liver Anoplocephalidae, and the most common nematodes.

The Role of Jackals in the Transmission of *Echinococcus granulosus* in the Turkana District of Kenya[1]

Calum N. L. Macpherson[2] and Lars Karstad[3]

The causative agent of hydatidosis in Kenya is *Echinococcus granulosus* (Batsch, 1786), which is primarily maintained in a cycle between dogs and domestic livestock (Nelson and Rausch 1963). Recent evidence suggests however that in Masailand, in addition to the domestic cycle, a sylvatic cycle is also operating (Sachs and Sachs 1968; Dinnik and Sachs 1972; Eugster 1978).

Jackals, being scavengers, are attracted to kills made by other carnivores and have every opportunity of becoming involved in a sylvatic cycle. In addition to preying on rodents, birds, and young and small antelopes, especially dik-dik, jackals also frequent the vicinities of manyattas and stock bomas, where they have access to the carcasses of domestic livestock (cattle, sheep, goats, camels, and donkeys), all of which may harbour hydatid cysts. In our experience, silver-backed jackals often feed on larger herbivores, as judged by the presence of bones, skin, and hair in their stomachs; whereas, the stomachs of the golden jackals examined in Turkana often contained mainly insects, including many scorpions.

Jackals have been reported as harbouring the adult parasite (Table 1); therefore, we decided to investigate the role played by the silver-backed jackal (*Thos mesomelas*) and the golden jackal (*Thos aureus*) in the transmission of *Echinococcus* in Turkana District. This district has the highest human incidence rate of the disease in the world (Wray 1958; Nelson and Rausch 1963; Schwabe 1969; Roettcher 1973; O'Leary 1976; African Medical and Research Foundation 1978–80).

The jackals examined for this study came from three separate locations in the north of Turkana District, near Kakuma, Lakonkai, and Lokichogio. In this area, annual morbidity due to hydatidosis is approximately 20 per 10 000 (African Medical Research Foundation 1978–80). This area was also known to support a small population of wild animals.

Four silver-backed jackals were captured from the Loita plains, Narok District, to examine the suitability of this carnivore as a definitive host for the parasite.

The jackals examined in Turkana were usually shot at night using a spotlight. The small intestines were then removed in toto, placed into labelled plastic bags, and stored in a refrigerator overnight.

After rinsing the intestine in normal saline, the gut was opened under fresh saline in large black-bottomed trays. If no *Echinococcus* were observed in this initial inspection, the mucosa was scraped and the scraping was washed and decanted several times with fresh saline. If *Echinococcus* were observed in the washings, they were placed in a petri dish of saline and allowed to relax for up to an hour. The number of worms recovered was estimated and the worms were fixed in either 70% alcohol, as recommended by Vogel (1957), or in 10% formol saline. Objects, which appeared on gross inspection to be either whole worms or segments of *Echinococcus*, were kept for microscopic examination. The specimens were stained in Gower's carmine and mounted in toto.

Experimental Infection of Silver-Backed Jackals

On the basis of finding infected silver-backed jackals in Turkana and reported infections from elsewhere (Nelson and Rausch 1963; Verster and Collins 1966; Eugster 1978), we artificially infected

[1]This paper was introduced by a well-illustrated oral presentation by Dr I. Mann, FAO/UNEP/WHO, Box 20360, Nairobi, Kenya.

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Table 1. Prevalence of *Echinococcus granulosus* in jackals in Africa (positives only).

Number examined	Positive		Country	Author
	Number	%		
Silver-backed jackal				
1[a]	1	100	Kenya	
9	1	11.1	Kenya	Nelson and Rausch (1963)
13	5	38.5	Kenya	Eugster (1978)
215	21	9.7	South Africa	Verster and Collins (1966)
Golden jackal				
82	1	1.2	Chad	Troncy and Graber (1969)

[a]Unidentified species.

four silver-backed jackals to further assess the suitability of these carnivores as definitive hosts for the parasite and to compare the infections produced in them with a control group of puppies.

The silver-backed jackals were captured from the Loita plains, Narok District. Prior to infection, each animal received 10 mg/kg body weight of praziquantel (Droncit (R), Bayer, Leverkusen, Germany). A few months later the jackals were fed a gelatin capsule containing 0.2 ml of packed protoscoleces obtained from hydatid cysts removed surgically from two Turkana patients. Prior to infection, the material was checked for viability by examining flame cell activity, evagination, and the uptake of vital stains. No material was used with an average viability of less than 60%. The same material was fed to five puppies, and all animals were examined post mortem 40 days later. Some of the worms obtained from the artificial infections were used for examining the isoenzyme glucose phosphate isomerase (GPI) employing the method recommended by McManus and Smyth (1979).

Results

Jackals Examined in Turkana

A total of 60 jackals were examined from the three locations in northern Turkana: of these, 28.3% were found to harbour the adult parasite (29% of the silver-backed jackals and 27.3% of the golden jackals). The golden jackals were obtained from Kakuma (1, negative), Lakonkai (12, negative), and Lokichogio (9, 6 positive). The silver-backed jackals originated from Kakuma (1, negative) and Lokichogio (37, 11 positive) (Table 2).

The morphological data obtained from the golden and silver-backed jackals were compared with material from naturally infected dogs from the same area. The material we collected corresponded closely with that of Nelson and Rausch's (1963), material that was collected in Kenya and accepted as being *Echinococcus granulosus*.

Table 2. Jackals examined for *Echinococcus* in Turkana.

	Silver-backed	Golden
No. animals examined	38	22
Light infection (<200 worms)	7	6
Medium infection (200–1000 worms)	1	0
Heavy infection (>1000 worms)	3	0

Experimental Infections of Silver-Backed Jackals

Unfortunately the results of this experiment were rather disappointing; only one of the jackals and one puppy became infected. However, both of these animals harboured very heavy infections, numbering some 4000 and 7000 worms, respectively. The location of the worms was similar in both animals, the small intestine being "furred" with worms from 54 to 72 cm back from the pyloris in the jackal and from 36 to 76.5 cm in the puppy.

The zymograms obtained for glucose phosphate isomerase (GPI) following isoelectric focusing of the soluble extracts of some of the worms from the experimental infections were found to be identical (Fig. 1). This suggests that the silver-backed jackal, as a different definitive host to the dog, does not appear to alter the electrophoretic pattern produced for this particular isoenzyme. Further experiments on jackals and puppies, using different isoenzymes, are required to confirm this preliminary observation.

Discussion

This is the first time that golden jackals have been recorded as definitive hosts of *E. granulosus* in Kenya. The first record of these animals harbouring

this parasite was made over 100 years ago by Panceri (1868) in Naples. Since then, natural infections in *T. aureus* have been reported from Palestine (Witenburg 1933), Pakistan (Lubinsky 1959), Algeria (D'Arces 1953), Sri Lanka (Dissanaike and Paramananthan 1960), in the Beka'a valley of Lebanon (Daily and Sweatman 1965), and in Chad by Troncy and Graber (1969), who found one infected jackal in the 82 they examined.

Although we found a high percentage (27.3%) of golden jackals harbouring *E. granulosus*, the greatest number of worms recovered from any one infection was 44, and the number of worms from the other five infections totaled only 22. In comparison to some of the silver-backed jackal infections, these were very light infestations indeed. No gravid segments were seen in the golden jackal material, although in other respects the worms showed normal development and all possessed testes. Daily and Sweatman (1965) reported that the single infected golden jackal they examined had two whole worms and three proglottids that were gravid, illustrating that although this was a low infection the parasite can achieve the gravid state in this host.

Of the 11 infected silver-backed jackals we discovered, three harboured in excess of 1000 parasites. The majority of the worms had gravid terminal segments, containing hundreds of shelled eggs, with the uterine lateral sacculations well developed. The first report of a natural infection in silver-backed jackals was by Nelson and Rausch (1963) who, in Kenya, found one of nine silver-backed jackals infected. In an extensive survey in South Africa, Verster and Collins (1966) found 21 of 215 (9.7%) silver-backed jackals to harbour the parasite and recently Eugster (1978) reported 5 of 13 (38.5%) infected in Kajiado District (Kenya). Three of the five positive animals reported by Eugster had worm burdens of greater than 20 individuals.

Viljoen (1937), working in South Africa, noted that silver-backed jackals could support the parasite and produced an experimental infection numbering a few thousand parasites. This, together with our own experimental observations, shows that the silver-backed jackal is a potentially good definitive host of *E. granulosus*.

The reasons as to why only one jackal and one puppy became infected are unclear, but the heavy infections produced in the jackal and puppy provide evidence that these animals are readily susceptible to infection with protoscoleces of human origin. This factor may have an important bearing in the epidemiology of the disease in Turkana and Masailand. The Masai only bury their dead when they have settled near townships, and the Turkana only bury respected old men and married women with children; others who die are simply left in the bush to be eaten by carnivores and scavenging birds such as vultures and Marabou storks. Wegener and Gathuma (1975) proved experimentally that the Marabou stork plays no part in the spread of *Echinococcus*. However, our experimental evidence shows that if either domestic dogs or silver-backed jackals were exposed to human hydatid cysts they could become infected. There is therefore the possibility that some of the infections found in these animals may have originated from the people themselves. This may be especially valid in Turkana District because the people have such an extraordinarily high prevalence of the disease. Therefore, in these areas man may not be the dead-end hosts, as in most other regions of the world. Undoubtedly the majority of the jackal and dog infections arise from scavenging of domestic livestock carcasses. There was ample evidence for this in Turkana after the recent drought. The skeletal remains of dead livestock, particularly cattle, were seen in thousands along all the roads and around the watering points.

The jackals may in turn be causing some infections by fouling waterholes used by the people and their livestock. Such waterholes are easily accessible to jackals, which visit them during the night.

One unusual route of transmission from jackals to humans is the eating of infected jackal intestines. The Turkana regard the intestines of most animals

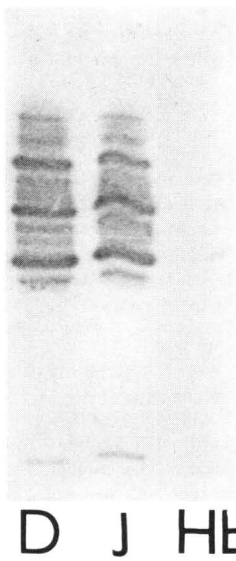

Fig. 1. Zymogram obtained from soluble extracts of E. granulosus tapeworms from a dog (D) and a silver-backed jackal (J) experimentally infected with protoscoleces from a Turkana patient, for the enzyme glucose phosphate isomerase (GPI). Hb represents a hemoglobin control.

as a great delicacy and would have consumed the small intestines of the jackals we had examined had they been permitted. However, they had to content themselves with the animals without that part of their anatomy.

Control

Recent studies have revealed that, in addition to the domestic cycle, there is strong evidence of a sylvatic cycle operating in Masailand (Sachs and Sachs 1968; Dinnik and Sachs 1972; Schiemann 1971; Eugster 1978; Macpherson et al. 1980). The presence of this sylvatic cycle will obviously complicate the planning of a control program for this area.

However, because the human incidence of the disease in Masailand is only 1-2 per 100 000 per year (Eugster 1978), such a program, although desirable, is not of such a high priority as it is in Turkana. In Turkana, jackals are the only wild animals to be found harbouring the disease. Of 154 wild herbivores (Grant's gazelles, warthogs, dikdiks, hares and squirrels) and 16 spotted hyenas examined in the District none were found to harbour the parasite (Macpherson et al. 1980). The main transmission of the parasite therefore is through a dog-livestock cycle. With the absence of a true sylvatic cycle, the problem of control of the disease in Turkana is much simplified and initial efforts for control should be aimed at the dog-livestock cycle. Reduction of the infection rate in domestic animals should suppress the jackal infection rate automatically, and no special measures of control need be applied to the wild animals.

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The Public Health Significance of Cysticercosis in African Game Animals

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Tapeworm cysts are frequently found in the muscles of slaughtered cattle in East Africa and are well recognized as being both a public health risk and a significant cause of financial loss to the meat industry. The cysts are almost always the larval form of the tapeworm *Taenia saginata*, the adult of which occurs only in the small intestine of man.

In an attempt to control the infection in man, cattle slaughtered in Kenya are inspected for muscle cysts and infected carcasses are further processed by freezing or cooking to ensure that the meat, when released, does not contain viable cysts. It has been estimated (Grindle 1978) that the annual loss from cysticercosis in cattle in Kenya is about £1 million. This estimate does not consider the inhibitory effect that cysticercosis has probably had on the development of a lucrative export trade for the beef industry.

Wild herbivores can also be infected with tapeworm larvae and it is probable that game animals slaughtered for human consumption would be liable to the same or similar meat inspection procedures as domestic livestock. It is a matter of some importance, therefore, to determine whether cysticercosis in game animals will jeopardize human health or be a significant restraint on the development of an industry based on cropping wild animals for human consumption.

Taenia saginata — "The Beef Tapeworm"

The earlier belief of many cattle owners that antelopes were a common alternative host for the cysticerci of *Taenia saginata* (*Cysticercus bovis*) has been shown to be erroneous (Nelson et al. 1965; Dinnik and Sachs 1969a; Woodford and Sachs 1973). There are only a few reports of *C. bovis* being found in wild animals and the risk of man acquiring *T. saginata* from eating game meat is probably very small. Nelson et al. (1965) found one wildebeest (*Connochaetes taurinus*) to have *T. saginata* cysticerci out of 92 wild herbivores of 19 species examined in Kenya. *C. bovis* has also been described in the buffalo (*Syncerus caffer*) in Angola (Sousa Diaz 1950) and in *Gazella dorcas*, *Gazella rufifrons*, and other antelope in Chad (Graber 1959). However, in large surveys of game animals in East Africa no evidence has yet been produced to show that wild herbivores are of significance in the epidemiology of *T. saginata*. In the Serengeti (Dinnik and Sachs 1969a) and in the Ruwenzori (formerly Queen Elizabeth) National Park in Uganda (Woodford and Sachs 1973) many of the animals examined were found to harbour muscle cysts but none was identified as *C. bovis*. In Kajiado District, Kenya, all the muscle cysticerci from several hundred wildebeest were species other than *C. bovis* (Chana 1975; Khalil et al. 1980).

C. bovis has been reported (Le Roux 1957) in an oribi (*Ourebia ourebi*) but this animal was tame and it is suggested it may have received massive numbers of eggs of *T. saginata* especially if its attendant was infected (Woodford and Sachs 1973). A Thomson's gazelle (*Gazella thomsonii*) that had been hand-reared from a few days of age in Kenya was found to be heavily infected with muscle cysts thought to be *C. bovis* when examined at 9 weeks of age (Karstad, personal communication). Specimens of cysts from the heart of this animal were examined by the authors and although the cysticerci were well developed no hooks could be seen on the rostella suggesting that they may have been *C. bovis*, but this could not be confirmed. Attempts to infect Thomson's gazelle artificially with *Taenia saginata* cysticerci have been unsuccessful (Fay 1972). The animals were, however, caught in the wild and dosed with eggs when between 4 and 6 months of

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age, by which time, resistance to infection may have been acquired. It can be extremely difficult in Kenya to infect domestic cattle of over 4 months of age with *C. bovis* and, to achieve frequent infection, eggs must be given in the first few weeks of life (Urquhart 1961; Froyd 1964). *C. bovis* has also been reported in giraffe kept in captivity (Mobius 1871; Schwartz 1928; Buckley 1947, 1948; Price 1961) but it is possible that the infection may have been acquired from human contact after the time of capture. It must be considered a possibility, therefore, that *C. bovis* could become more prevalent in game animals if these were to be reared under domestic conditions where close contact with infected humans could occur.

Taenia solium — "The Pork Tapeworm"

Taenia solium, the other *Taenia* tapeworm that infects man and develops to the adult stage in the small intestine is acquired by eating the cysticerci (*Cysticercus cellulosae*) that occur in the muscles of pigs. This tapeworm has a scolex bearing a double row of hooks and thus has some resemblance to the cysts found in wild animals in East Africa. The tapeworm has the ability to use man as an intermediate host and cysticerci can develop in the nervous tissue and can sometimes result in serious disease. It is therefore necessary to determine whether wild animals in Africa are infected with this parasite.

Cysts resembling, but not confirmed to be, those of *C. cellulosae* have been recorded in a bush pig (*Potamochoerus choeropotamus*) in Southern Africa (Viljoen 1937). *C. cellulosae* has been described in a warthog (*Phacochoerus aethiopicus*) (Hamerton 1947) but this animal died after 10 years' residence in London Zoo and may have acquired the infection while in captivity. However, in the survey carried out by Woodford and Sachs (1973) no muscle cysts were found in 106 free-living warthogs examined in Uganda. The larval stages of *T. solium* have been described by Verster (1969) from the vervet monkey (*Cercopithecus aethiops*), bushbaby (*Galago* sp.), and rock hyrax (*Procavia capensis*). Differentiation by morphological features of the muscle cysts that have been found in wild herbivores in East Africa has shown that they are a species other than *C. cellulosae*. At present, there is no reason to believe that game animals likely to be used as a source of meat for human consumption would be involved in the transmission of *T. solium* to man.

In Kenya, *T. solium* cysticerci have only rarely been reported in domestic pigs (Solomon 1932; Viljoen 1937; Anon. 1962, 1963) but they are more commonly recognized in other African countries (Merle 1958; Verster 1966). If there is an expansion of the pig industry, it is possible that *T. solium* may become more widespread in East African countries. In such a situation, the spread of the parasite to the wild animal population could not be ruled out.

Nelson et al. (1965) suggested that many of the cases of cysticercosis in man in Africa may have been caused not by *T. solium*, as generally assumed, but by *Taenia* spp. from dogs or wild carnivores. However, the few reports of cysticercosis in man in East African countries, where there is a large population with wild carnivores, suggest that if there is a risk of infection with wild animal cysts, it is very low.

Larval Tapeworms in Wild Animals

Whether it is completely safe to eat game meat infested with muscle cysticerci of species other than *C. bovis* and *C. cellulosae* is still an unanswered question. At present, however, the available circumstantial evidence would suggest that the cysts are not infective to man. There are no reports of adult tapeworms of game-animal cysticerci ever being found in man despite the fact that the flesh of wild animals has been and continues to be frequently eaten by a large section of the human population in Africa. This is not unexpected because adult *Taenia* tapeworms exhibit a fairly high degree of host specificity, more so than do their larval stages. For example, no adult tapeworms of hyena have been recorded from lion although they eat the same prey. Immature tapeworms resembling those from the hyena and hunting dog have been found in lions but it was suggested that they are unable to reach maturity in a host seemingly unsuitable to them (Dinnik and Sachs 1972).

Although many of the cysticerci found in wild animals in Africa cannot yet be positively identified, several species have now been recognized. Adult tapeworms recovered from wild and domestic carnivores in Africa correspond in morphological features such as hook size, number, and shape with certain cysticerci in wild herbivores (Table 1). On this basis, it has been concluded that the larval and adult tapeworm are the same species and in a number of cases this has been supported by experimental infection of wild animals (Verster 1969).

Not all the cysts are found in the musculature of the host. Serosal cysticercosis is frequently encountered in game animals (Sachs 1969), the cysticerci either being attached to serosal surfaces or lying free in the peritoneal and pleural cavities. There is no

Table 1. Species of *Taenia* occurring in carnivores with larval stages in African game animals.[a]

Tapeworm	Final host	Common site of larval tapeworm
T. regis	Lion, leopard	Serosa in peritoneal and pleural cavities
T. simbae	Lion	Serosa in peritoneal and pleural cavities
T. gonyamai	Lion, cheetah	Muscle
T. crocutae	Spotted hyena, Brown hyena	Muscle
T. hyaenae	Spotted hyena, Brown hyena, Hunting dog	Muscle
T. olngojinei	Spotted hyena	Epidural space of sacrum
T. acinonyxi	Leopard, cheetah	Muscle
T. hydatigena	Dog, silver-backed jackal and other canines	Serosa in peritoneal cavity
T. multiceps	Dog and other canines	Brain, spinal cord

[a]Data compiled from Verster (1969) and authors' (L.F.K. and A.J.) own investigations.

evidence that they are infective to man although there is still debate as to the identity of many of these cysts. In parallel with the procedure in domestic livestock, in a heavily infected animal it may be considered necessary to condemn the offal at meat inspection.

Coenuri, the large cysts of *Taenia multiceps*, can also occur in African mammals and are not always found in the central nervous system. Verster and Bezuidenhout (1972) recovered a coenurus from the hindquarters of a gemsbok (*Oryx gazella*). The cyst was fed to a domestic dog and subsequently many adult *T. multiceps* were recovered from its small intestine. Again there is no evidence that man can become infected by eating meat harbouring this tapeworm.

A curious parasite has been described in certain East African antelopes. Wildebeest, hartebeest (*Alcelaphus buselaphus*), and topi (*Damaliscus korrigum*) have frequently been found to harbour cysticerci within the epidural space of the sacrum (Dinnik and Sachs 1969b; Woodford and Sachs 1973; Khalil et al. 1980). These are the larval form of *Taenia olngojinei* found in the spotted hyena (*Crocuta crocuta*), an animal with jaws strong enough to crack the sacrum and thus ingest the cysticerci.

The Effect of Cysticercosis on the Utilization of Game Meat

Meat infested with cysticerci is condemned primarily because of the associated risk of infection of man. However, even if it could be definitely established that game animal cysticerci are not a risk to man, heavily infected carcasses would not be sold because of the appearance of the meat. Dinnik and Sachs (1969a) describe one impala (*Aepyceros melampus*) they examined in the Serengeti area that had an extremely heavy infection and 165 muscle cysts were found in 0.5 kg of the *musculus biceps femoris* and *musculus semitendinosus*. However, they note that such a heavy infestation was seldom observed and generally only a few cysticerci were found in infected carcasses.

There is no doubt that the prevalence of infection of game animals with muscle cysticerci can vary markedly. For instance, Thomson's gazelles were found to be frequently infected with cysticercosis in Kenya (Fay 1971). In contrast, the same species of gazelle in the Serengeti region of Tanzania was seldom infected; whereas, the closely related Grant's gazelle (*Gazella granti*) in the same location was commonly heavily infested (Sachs and Sachs 1968). There is a definite need for research into the factors that may be involved in the prevalence rates of cysticercosis in game animals.

High prevalence rates of infection have been recorded in a number of antelope species in East Africa. In the Ruwenzori National Park, 8 of 11 bushbuck (*Tragelaphus scriptus*) and 11 of 14 reedbuck (*Redunca redunca*) harboured muscle cysts (Woodford and Sachs 1973). In the Serengeti, 60-80% of wildebeest, topi, kongoni (*Alcelaphus buselaphus cokii*), dik-dik (*Rhynchotragus kirki*), and Grant's gazelle were found to be infected (Sachs 1966). In Turkana District of Northern Kenya, 8 of 10 dik-dik had muscle cysts, the number in each carcass ranging from 1 to 14. In contrast, in 11

Grant's gazelles from the same region examined at the same time, only one live cyst was recovered from five animals that were totally dissected and one calcified cyst was recovered from the other six gazelles that were examined by multiple incisions into the musculature (Stevenson and Karstad, unpublished).

From the results of a pilot scheme carried out in the Serengeti area, it was estimated that the game meat liable to be rejected as a result of parasite infestation would be equivalent to 15–20% of the total carcass yield (Schindler et al. 1969). To avoid the loss of much valuable protein, it was recommended that the meat be canned and sterilized before being marketed (Glees et al. 1972). However, as pointed out by Woodford and Sachs (1973), the high proportion of herbivorous animals found to harbour muscle cysts in the Serengeti and the Ruwenzori National Park may not reflect the situation in game animals on farms or in hunting areas where the predator population may not be as large. A careful examination of the level of infection in a sample of animals is needed before any decision is made to crop a game animal population to provide meat for human consumption.

Of the 555 wildebeest examined by Khalil et al. (1980) from Kajiado District, Kenya, 59% were found to harbour muscle cysts. However, although the prevalence of infection would appear to be high, the level of infestation is generally low in wildebeest from this region (Chana 1975). If such a population was to be cropped to provide meat for human consumption, a decision would have to be made as to what treatment, if any, a lightly infected carcass should undergo.

The meat inspection rules laid down for the examination of carcasses of domestic cattle should not necessarily be applied to game animals at slaughter. The lack of evidence, to date, of a public health risk being associated with the cysticerci found in wild herbivores in Africa suggests that it may be unnecessary to reject as unfit for human consumption carcasses that are lightly infected with muscle cysts. However, there is undoubtedly a need for further research into the identity, life cycle, and infectivity of wild animal tapeworms. Unfortunately, final confirmation of the harmlessness to man of the muscle cysticerci in game animals must depend on the demonstration that ingestion of live cysts by human volunteers does not lead to the development of intestinal tapeworms.

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The Value of Research Findings to the Research Director

S. Chema[1]

Wild animals have been implicated as carriers and disseminators of diseases for a very long time. However, only recently have we, in Kenya, felt we were making some progress in defining the true roles of wild animals in the epidemiology of diseases such as foot-and-mouth disease, rinderpest, and East Coast fever. The research director often finds himself in the position of an officer who cannot always be at the battlefront. He does not receive day-to-day bulletins of successes and failures in research on all fronts; therefore, workshops such as this are extremely informative and useful.

Wild animals have often been used as a scapegoat for the variety of disease problems that from time to time beset domestic animals. Some of the complaints against game have been justified (malignant catarrhal fever and corridor disease). Many others have been speculative and unfounded. Research is essential to provide plausible and satisfactory answers to these questions. Earlier we were told that the buffalo is the only wild animal found, so far, to be a frequent carrier of foot-and-mouth disease in Kenya, but we were also cautioned against overemphasizing its role as a reservoir for infection of cattle because transmission trials from carrier buffalo to susceptible cattle have all been unsuccessful. We learned that while rinderpest was at first a great killer of wild ruminants and pigs, it has ceased to be so, and, in the face of an adequate program of vaccination of cattle, rinderpest in wildlife has disappeared. It was pointed out that it may in fact be the domestic animals that are the menace to wildlife. Although it is no longer visible as a killer disease, rinderpest may not be gone because recent serologic surveys have found antibodies to rinderpest virus in healthy wild animals. We must therefore be on the watch for wild strains of rinderpest virus persisting in wildlife populations. We wonder if these mild strains might sometimes revert to virulence.

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Malignant catarrhal fever (MCF) is now quite well defined. However, unfortunately, we have not yet achieved our goal of protecting the Masai cattle from contracting this disease from wildebeest.

Rabies, on the other hand, is an example of a problem that, in Kenya, requires much more definition. Research is needed to determine if there is a significant wildlife reservoir, to identify the most important species involved, and to learn, by the experimental approach, the responses of these wild animals to infection with rabies virus — especially their ability to transmit the virus. All of this information must be provided by the researchers before we can make a more rational approach to rabies control. With rabies we must keep in mind the possibility that the rabid wild animal may be only the indicator species, while the important long-term reservoir may be an animal in which the virus does not produce such dramatic effects.

Our researchers who work on the diseases of wild animals have cautioned us to be specific, not to blame all wildlife for the sins of a single species. For example, of the several wild ruminant species examined in areas where bovine petechial fever occurs, only the bushbuck was found to carry the rickettsial agent. Even then, we have no proof that bushbucks serve as reservoirs for infection of cattle. Our knowledge is far from complete.

In theileriosis (East Coast fever), only the buffalo, among wild animals, has been found to carry a *Theileria* pathogenic for cattle. Researchers have been hopeful that the wild ruminants that have learned to cope with their theilerial parasites may provide clues to methods for immunizing our cattle against ECF.

Research on trypanotolerant wild animals also may guide us to a solution to the problem of trypanosomiasis in domestic ruminants. What we are saying is that we would like to learn the secrets of how the wild animals have learned to tolerate the parasites that are so destructive in our domestic animals. I have a suspicion that one of the other speakers is going to say: Why bother? Why not just raise the

wild animals instead of cattle, sheep, and goats? I am sure most people will watch his progress with great interest.

Research on echinococcosis in Turkana has given us more hope that a control program may succeed. The apparent absence of a sylvatic cycle in Turkana should simplify, somewhat, a very difficult and complex disease control problem.

In conclusion, let me say that research on diseases of wild animals is regarded as a valuable and necessary adjunct to our other veterinary research programs. Because it crosses government organizational lines, we could argue that a joint research effort, perhaps in a jointly supported research facility, should be developed to serve the interests of both the Ministry of Livestock Development and the Ministry of Environment and Natural Resources. The attendance of a number of people from both Ministries, as well as the papers presented by workers from a variety of outside laboratories or organizations, are evidence of a healthy multifaceted approach to problems facing the cohabitation of wild and domestic animals. It is an approach that I intend to promote and encourage in all our research programs.

The Role of Wildlife Disease Research in Livestock Production

L.J. Howard[1]

It is true that the livestock producer, whether rancher or farmer, has laid considerable blame on the presence of wildlife for sudden outbreaks of disease in his domestic stock. Disease lies second only to nutrition in the possible success or failure of a livestock enterprise. The farmer's greatest fear is one of disease, quarantines, and restrictions, which prevent marketing of stock at an opportune time, increase overheads, reduce grazing and therefore reduce margins of profit. The fear of quarantine restrictions is so real, that farmers and ranchers have been known to muddle through quite severe outbreaks of disease without requesting the help of local veterinary officers. When asked why, they will reply: "The Vets! All they are looking for is an excuse to put you in quarantine!"

Fencing against incursions of game is almost economically impossible on properties bordering natural reservoirs of wildlife. The natural steeplechaser of the wildlife world, the eland, will take a normal five strand paddock fence in its stride, as the eland herd moves from paddock to paddock. Eland have also been observed negotiating a 6-ft (1.8-m) high chain link fence, although where others had jumped before, the fence had sagged to 5 ft 7 inches (1.7m) at the position of the jump. Kongoni, a little more cautious and sedate, will seek a gateway or other opening before attempting to jump, but when disturbed, crash through fences as if nothing barred their way. Even the ostrich has been observed running at 24 kph straight at a five strand boundary fence; its own impetus somersaulted the bird over the fence, broke two top strands, and landed the bird in a heap of feathers and legs on the opposite side. The Thomson's gazelle will squeeze through an 8 inch (20 cm) gap between the bottom two strands of wire. All told, it is almost impossible to exclude game using normal or standard fencing practice.

On occasions of seasonal incursions of wildlife onto properties bordering natural wildlife reservoirs, I have often observed up to 400 kongoni, 150 eland, and 300 wildebeest concentrated in an area of no more than 3000–4000 acres (1200–1600 ha). In these circumstances the buildup of ectoparasites and, possibly to a lesser extent, endoparasites is enormous, and there is considerable risk from the increase of ticks, which may well be vectors of some of the common tick-borne diseases that affect domestic livestock. In certain circumstances, or perhaps extraordinary circumstances, the increase in the tick burden is so great that only two-day dipping in an organophosphate acaricide will control the invasion. In the absence of *R. appendiculatus* the disease risk is notably less, but in wetter tick habitats there is considerable risk of the introduction of disease vectors.

There is no doubt that there is sometimes a correlation between seasonal incursions of wildlife and an increase in the incidence of tick-borne diseases, but this is entirely due to an abnormal increase in vector challenge or to normal tick control procedures being inadequate to maintain control over an exceedingly abnormal challenge. Fortunately, research into the correlation between wildlife diseases and those of domestic stock has taught the livestock producer that there is little risk of pathogenic tick-borne organisms being transmitted to domestic livestock, and that generally, wildlife are not reservoirs of diseases that are likely to bring ruin to the producer overnight.

In July 1977, I was offered the opportunity to become involved in the management of Galana Game and Ranching Ltd., situated in the coast hinterland approximately 60 miles (100 km) inland, where an opportunity arose to put research theories into practice. The ranch was a large-scale enterprise covering 5000 km^2 (approximately 1.5 million acres). Previous aerial censuses gave estimated wildlife populations at one time as 5000 elephant, 7000 fringe-eared oryx, 50 black rhinoceros, 3000 buffalo, 3000 zebra, 1500 eland, and 400 giraffe. Smaller ungulates in undetermined numbers also shared the habitat, such as Peters' gazelle, waterbuck, impala, lesser kudu, topi, gerenuk, hartebeest, duiker, and

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dik-dik. Daily maximum shade temperatures averaged 34-35 °C with humidity rising to 90% during parts of the year. Rainfall in this semi-arid bush area of the coast hinterland averaged approximately 500 mm (20 inches) in the east to a low of 125 mm (5 inches) in the western desert-scrub area of the ranch. The domestic stock comprised, on average over the period under review, between 15 000 and 20 000 head of Boran cattle, 1500 sheep and goats, 100 camels, 100 domesticated oryx *(O. beisa)*, and 20 or so domesticated eland. The ranch was situated in an endemic trypanosomiasis area and heavy rainfall during the 2-year period enhanced tsetse fly dispersal of the four species of *Glossina* identified in the area, namely *G. pallidipes*, *G. longipennis* *G. austeni*, and *G. brevipalpis*. The extraordinary wet conditions also increased the challenge from bacterial diseases previously encountered; *Salmonella*, *E. coli*, and *Pasteurella* infections in calves and young stock. The developed area of 640 000 acres (256 000 ha) was adequately served by five sprayraces and well-watered from riverside and inland boreholes. To rest the dry weather grazing in the developed area, the cattle and in fact all stock, were moved north during the rains, utilizing surface water and one dam. No tick control facilities were available in this area, which was grazed for three to four months of the year. In view of this, there appeared to be little point in maintaining a high standard of tick control when cattle were grazing in the developed area because the stock were exposed to challenge when grazing in the undeveloped area in the north of the ranch.

At that time, little was known by the management of the risks involved in exposing domestic stock to challenge from tick-borne parasites hosted by wild ungulates. Although it had been known for some time that buffalo were reservoirs of *T. lawrencei*, it was considered, because random tick samples forwarded for identification had not revealed the presence of *R. appendiculatus*, that the resident buffalo could well be free of this particular pathogen. Later the Wildlife Diseases Section was able to confirm that this was in fact the case. Information, however, was also required on the effects of other parasites carried by wild ungulates, piroplasms, *Anaplasma*, and *Babesia*. Wildlife disease research data were perused to ascertain the degree of tolerance our domestic stock might have to these parasites, and with the exception of the possibility of a pathogenic strain of *T. mutans*, the risk was apparently very low. Some experience had already been observed in respect of the latter parasite when a steer died of theileriosis during the time that stock were grazing in the eastern area, a normal buffalo habitat. Laboratory reports indicated that the *Theileria* was probably *T. mutans* and that the animal had recently recovered from a *T. congolense* infection. In the light of this information it was considered that the *Theileria* infection was most likely a premunity breakdown occasioned by the stress from the trypanosomiasis infection. Although it was appreciated, from research data, that wild ruminants were capable of maintaining a reservoir of the rickettsial agent of heartwater, it was considered that, because young calves appeared to have a strong natural immunity, exposure to a natural challenge from birth might result in the calves becoming infected while they still retain a partial maternal passive immunity and so become immune themselves. This is an instance where the results of wildlife disease research influenced the decision to deliberately encourage the development of a state of premunity and immunity against tick-borne diseases in domestic livestock ranched within a natural wildlife reservoir.

Blood smears taken from the cattle to ascertain the incidence of trypanosomiasis revealed that the six-monthly prophylaxis cover against the disease using Samorin at 0.25 mg/kg body weight was not having the desired effect. Wildlife research papers revealed that certain species of wildlife, namely giraffe and buffalo, were proven carriers of trypanosomes although they themselves remained healthy.

The fact that some of the resident wildlife were carriers of the disease was rather immaterial because we were ranching in an endemic area where blood slides had shown that the domestic stock were also carriers. It was obvious that the recurring breakthrough of the prophylaxis was occasioned not by the presence of wildlife carriers, but by the movement of stock through and to areas of varying fly challenge. The dosage rate of Samorin was increased to 0.50 mg/kg body weight quarterly, with excellent results.

The increased cost of trypanocidal drugs resulted in the need for savings. At the time, all the cattle were vaccinated at six-monthly intervals using a quadrivalent foot-and-mouth disease vaccine. We were ranching in a fairly isolated area but, nevertheless, it was difficult to evaluate the immunity provided by this vaccine in a wildlife reservoir such as Galana. Wildlife disease research into the role played by wildlife in the spread of foot-and-mouth disease was studied. The information conveyed by these studies influenced the decision to withdraw the policy of regular vaccination. Close liaison with the Provincial Veterinary Officer was maintained regarding outbreaks in the surrounding district and vaccination cover was provided when outbreaks or quarantine boundaries were within 30 miles (50 km) of the ranch boundary. Only one vaccination cover was given and no outbreaks occurred on the ranch during the two-year period under review.

The reduction in the mortality achieved by this disease control policy, which was only considered after detailed perusal of research information, serves to indicate the possibilities available to the mixed wildlife–domestic stock producer. Mortality decreased from 3.5% in 1976–77 to 2.61% in 1977–78 to 1.99% in 1978–79. (Rainfall during these periods was: 11.75 inches, 19.5 inches, and 30.0 inches.)

Without research into livestock disease the livestock industry of this country would not have developed to maturity. Although wildlife disease would appear a lesser consideration, no livestock development program is able to progress to its full potential without veterinary research.

Research into wildlife diseases, and interrelation of these diseases with domestic stock, has not only provided information that assists in the formulation of management policies but has confirmed the presence of a comparatively disease-free source of animal products. If the commercial exploitation of Kenya's rich reservoir of natural fauna is to be encouraged, development in this direction must include veterinary research facilities.

Very little is known of nutritional diseases in wild ruminants and these diseases may well become apparent under confinement or domestication. Current research has not reassured the potential wildlife producer that close collateral development of wild ruminants with domestic stock will not result, within a few generations, in host-specific strains of protozoa becoming interadapted or even pathogenic to their new hosts. Is not *T. lawrencei* an adapted strain of *T. parva*? Why has the hitherto nonpathogenic *T. mutans* suddenly produced a pathogenic variant strain? Foot-and-mouth disease virus, noted for variant strains, may well become an immunological problem in collateral development of species.

Research must continue into methods of immunization of domestic livestock against those diseases of economic importance that seriously affect the economy of ranchers and farmers operating in specific habitat zones. Until success is achieved in these fields, livestock producers will be unable to accept the general approach that wildlife are of little importance to the incidence of diseases affecting domestic livestock.

In addition to the comparative immunity to disease shown by wild ungulates, a variety of fauna are able to maintain productivity in areas unsuitable or marginal to domestic stock because of their ability to make use of a much wider range of vegetation. Because of various physiological adaptions, some species appear able to obtain their liquid requirements from the herbage and are hence almost independent of water. Large areas of unproductive land situated in tsetse-infested and semi-arid areas now lie within the reach of economic production. One such development, believed to be the first collateral enterprise of cattle/domesticated oryx ranching combined with wildlife management through tourism and hunting, was the formation of the local company Galana Game and Ranching Ltd.

With the cooperation of the Game Department, wild oryx (*Oryx beisa callottis*) were captured. Several studies sponsored by the African Wildlife Leadership Foundation have shown that this ruminant, which is suited to arid rangeland, can be domesticated and ranched under certain management techniques. Further studies have shown that the oryx is able to digest protein and crude fibre significantly better than cattle, due possibly to a faster rate of high fibre food fermentation in the rumen. Adjusted for body-size difference, the oryx has a water requirement that is only 15–20% of that of a cow. This factor, combined with the oryx's high heat tolerance, makes it the ideal meat animal for the area.

The Wildlife Diseases Section at Kabete has been associated with the oryx project for some time. Intensive disease studies have been made on the oryx. Possibly the most significant finding is that, in addition to the oryx's resistance to *T. congolense* infection, the animals exhibit aggressive behaviour toward tsetse flies that gives little opportunity for infection to take place. These findings further suggest that the oryx is the most suitable species for meat production in endemic trypanosomiasis areas. There is sufficient information on the oryx to enable the project to explore the possibilities of economic production; however, restrictions on marketing and on catching wild oryx for domestication, which is essential to build up herds to an economic unit, make this impossible.

No more than 5 years ago Galana operated a very successful tourist and hunting operation controlled by a resident hunter and a wildlife conservationist of the highest esteem. With the liaison, cooperation, and sincere interest of the Game Department, annual quotas were allocated for each species of game animal. During the fiscal year 1974–75, of the 511 animal quotas allocated, only 236 were utilized due to the very high standard of selection insisted upon by the resident hunter and the company. Thirty-nine of the animals taken by overseas visiting sportsmen qualified for the Rowland Ward Book of Records and one Peters' gazelle gained the honour for the world record head. Ivory from the 13 elephants taken by sportsmen averaged 67 lb (30.38 kg), the heaviest tusk weighing 101 lb (45.8 kg). Such records are only maintained through a planned long-term policy to improve the quality of the fauna.

A full-time motorized antipoaching unit, manned by seven armed private game scouts, was operated at an annual expense in the region of £3000 per annum. During the year reviewed, 31 poachers were

arrested, of which 29 were convicted of poaching. During the succeeding years, the sale of ivory was banned followed by the banning of hunting. This resulted in the closure of the safari business and sadly, due to the lack of income, the disbanding of the antipoaching unit. The resident antipoaching team was not replaced by the Government, whose facilities were already stretched to the limit. Armed poachers moved in to the extent that certain species are in danger of becoming extinct and trophies of the standard recorded in 1974–75 may never be seen again in our lifetime, certainly not of the elephant.

The last two paragraphs serve to illustrate that when the fauna of this country is offered to private enterprise for commercial exploitation the conservation of wildlife is ensured. When that responsibility is withdrawn, the whole consensus of conservation is endangered.

Wildlife Ranching in Perspective

David Hopcraft[1]

It is necessary to put the concept of wildlife ranching in its true perspective prior to looking to its future or to its associated problems. From 15 years work, it is evident to me that the utilization of wildlife for meat and hide production is the only alternative we have to our present systems of domestic stock ranching. And more importantly, it is a land-use system that by its nature is nondestructive. It is part of the African ecosystem and, as such, is the key to the survival of those seriously threatened degrading lands.

Desertification is the new word that we use to describe land that has reached a serious stage of deterioration. It spells desperation, starvation, and death. The official figures relating to the process are terrifying. Ninety-five percent of all arid and semi-arid grasslands of Africa are threatened. This covers 45% of the remaining four-fifths of the non-desert mass of Africa: three-quarters of Kenya is semi-arid or arid grassland.

It is imperative that we understand this process, which undoubtedly is the biggest threat to our continent. It is not advancing sand covering up grasslands. Rather, it is the gradual degradation of land, loss of grasses followed by wind and water erosion of the soils themselves. It is not a matter of less rainfall, but rather the runoff of rain, which causes erosion and effectively reduces the amount of water absorbed. The process is a man-made phenomenon. It is the end result of human intervention in a natural system.

We start from a natural ecosystem: delicate soils; healthy vegetation; very little, if any, water; yet teaming with a large variety of animal species — each living in harmony with the whole. We see man living within and from this unity, a component of the whole. Then comes the movement of man into the modern era — he now wishes to control, and develops the means to do so.

He has domesticated certain animal species in the temperate zones and has become used to them. They, for him, become the means of food production from grasslands. As he spreads his new knowledge into our drier areas, he brings his animals along.

In the rush to develop, to bring progress, man has given no thought to what nature evolved, the equally efficient members of — certainly in Africa — the same family as his domesticated cattle. These are brushed aside. And so, the ecosystem, so carefully evolved and balanced, is violently attacked, altered, and yet expected to continue in all other respects the same under this new regimen of cattle, sheep, and goats. To make matters worse, overstocking occurs. But the alteration in animal type is immediately felt by the rest of the environment. Instead of a multiculture, maybe 10–20 ungulates, we now have monoculture — only cattle. Certain grass species are naturally preferred by this new animal and are grazed out; a second line is then attacked. Gone is the utilization of the whole vegetational spectrum. The balance is upset. Insidious down-grading changes begin to occur. Species change, which is soon followed by a reduction of cover, for to make matters worse, this imported cow needs water, and must walk to it, tracking and trampling the land to get there. Erosion sets in along the tracks; compaction occurs, reducing the water absorption and making the soil drier; vegetation is reduced and dries out more quickly. Bare patches occur and the soil is heated by the sun. Overall, we see the process of desertification in full swing.

The Wildlife Land-Use Alternative

The question is whether we could utilize wildlife, Africa's original grazing animals, to lead us back, or should I say forward, to a healthy productive system. They were part of the original system that was biologically rich and stable. Could they be the key to the riddle of desertification?

In collecting data worldwide on the problem of land degradation, I came to a startling conclusion. It was only where humans had eliminated the natural

[1]Wildlife Ranching and Research, Athi River, Kenya.

game herds to make way for their cattle, sheep, and goats that the process occurred. Where the natural herds were left alone, either in National Parks or in tsetse areas, no land degradation occurred. The examples stretch from the dust bowl of the American west, across Asia, to Africa.

A variety of means and methods have been tried to use the wild ungulates. However, these have all been half-measures — either ranchers using game as supplementary income to their "stock," or projects, both business and scientific, to "crop" these animals. No management has been carried out, for this implies control of both the land and animal resources. It is a strange fact that no full-scale game ranches have been tried. Most African countries have not even contemplated the idea. Yet, most scientists would agree that a variety of game animals ranched on a given piece of land would produce more meat and hides per hectare than the use of cattle. Coupled with less cost, and a nondeteriorating grassland system, surely we should at least try this system.

Starting in 1965, with a grant from the U.S. National Science Foundation, a study was carried out to look into this option and determine the viability of even one wild game species in comparison with cattle. Through several years of actual ground trial and measurements, some startling results were uncovered. These results give insights into several aspects of land use: (1) ecological, in terms of survival of the land and vegetation; (2) secondary production per unit area (i.e. meat produced per acre); and (3) financial, in terms of returns per unit area, both gross and net.

Ecological

The area used was homogeneous in terms of vegetation and had similar slope and soil types across the area. The enclosures were adjacent. The effects on this range of the two species were markedly dissimilar (Table 1).

It is clear from Table 1 that cattle adversely affect this type of grassland. Each year the situation becomes worse and in many African countries, this same deleterious progression has led, and is continuing to lead, to disaster.

Secondary Production

The natural increase of weight within each of the enclosures was carefully measured. Carcass and lean-meat measurements were taken. The lean-meat figures, expressed as pounds per acre per year, were: experimental gazelle 14.6; experimental cattle 7.9; average ranch cattle 4; and traditional stock 1 (1 lb/acre equals 1.12 kg/ha). These data give an indication of the potential of one species of animal within in its natural area and indicate that adaptation to the environment is very important. These animals expend far less energy than imported animals to overcome the harsh elements of vegetation, disease, temperature, and weather conditions. Thus, more energy is available for growth.

Financial

In assessing the income generated, we take into consideration meat, hides, and cost. The meat prices for the wildlife that has been cropped in most parts of Africa have been higher than beef. In West Africa, this price is usually double. However, even taken at equivalent prices, we can count on 50–100% more meat to sell per acre.

Hides command a very good price. During the trials a gazelle hide was fetching $5; a cow hide brought less than $4. A 10-acre (4-ha) area was needed per cow and it took 3 years to produce a hide. Eight gazelles were turned over per year in the same area, giving 24 hides. Thus, the income was $120 to $4 — 30 times more.

A much greater gross income is generated from the gazelle. If the whole natural spectrum of game animals was used, that income would be higher still, as each species lives within a different niche on the range, and thus the overall carrying capacity of the land is improved.

Without looking at costs, we would therefore see a substantially higher gross income from the indigenous animal. Costs for these animals, however, are negligible because no dipping, inoculations, water supplies, night enclosures, etc. are required. Our present estimates are that costs on a cattle ranch run about 66% and on a game ranch about 20%. Whichever way the above picture works out, it is clear that the natural environment is an extremely wealthy resource. The question is how to reap this wealth.

The Present Game Ranching Project

The first full-scale demonstration game ranch, established to attempt putting these ideas into practice, is on 20 000 acres (8000 ha) within 25 miles (40 km) of Nairobi. The area has been perimeter-fenced and game numbers have increased from 900 to 7000 in 2.5 years. Cattle on the ranch are in the process of being phased out, demonstrating a gradual conversion to game. The idea is simply to run such a conversion on a financial basis. As the income is obtained from the game, the cattle numbers are diminished.

Table 1. Effects of cattle and gazelle on the same rangeland.

Item measured	Cattle enclosure	Gazelle enclosure
Cover	Significantly reduced	Not reduced (32% more cover than in cattle enclosure)
Species	Reduction in climax vegetation, particularly *Themeda triandra*	100% more climax grass species than found in cattle enclosure
Tracking	Significant, particularly to water trough	None
Devastation around water trough	Serious and extensive	None

In 1976 a loan was obtained from the World Bank through the Agricultural Finance Corporation and a grant for the initial conversions and research was obtained. This came from the Lilly Endowment, after nearly 2 years of visiting nearly all of the major funding agencies in the U.S. The project was accepted by and has been operated through Cornell University.

Problems Facing Wildlife Utilization

Emotional

Because of the catastrophic destruction of wildlife, estimated at between 95 and 99% in Africa, wildlife utilization has become a very emotional issue. This problem, in conjunction with sentimental attitudes, has significantly increased the influence of many organizations that rightly exert pressure to try to prevent further loss of animals.

Unfortunately this is too often done without the realization that the land and animals are indivisible. Land has a capacity to support a set number of grazing animals. If they be cattle then the wildlife must go. This is the continuing trend. It is only when we begin to understand the desirability of game animals as an ecological and productive alternative that true conservation will be effected.

I quote here from the writings of President Roosevelt following his safari to Kenya in 1914. "When genuinely protected, birds and mammals increase so rapidly, it becomes imperative to cull them. The foolish sentimentalists who do not see this are the really efficient foes of wildlife, and of sensible movements for its preservation."

Political

Despite the fact that we were given permission by the Government in 1975 to operate a game ranch, we have for the last 2 years been waiting for the licences to operate. This has thrown the operation into confusion and near bankruptcy. Policies handed down to the newly independent Government of Kenya were based on many years of negative thinking and control. They came from the the 17th century concepts of control and prevention of anyone taking game. The animals were declared "King's game" or "Royal game," and anybody found "utilizing" was a poacher. Thus antipoaching was founded. And "anti" anything to do with game is our legacy.

I have in the last 8 months worked with the wildlife department and the Ministry attempting to obtain a change in policy. It is happening, and the Minister of the Environment, in a letter to Cornell, has agreed to the wildlife ranching program and committed himself to issuing the necessary permits.

The emotional and political problems in combination have been all but insurmountable. In logical terms, it must be one of the strangest aspects of our beliefs and policies: it is acceptable, and we are allowed to use Asian and European animals in Africa, but it is not acceptable to use African animals.

Biological and Veterinary

Because of the above problems very little attention has been paid to African animals by the scientific community. There are research stations, universities, and facilities to study domestic stock all across Africa, but knowledge of wildlife is woefully lacking.

This creates enormous problems for the development of any game utilization projects. From the management point of view there is very little data

available on which to base any decision-making process. Even the simplest things such as growth curves, aging criteria, weight gains, and population data are sketchy and inaccurate. Health data, from normal parameters to any disease conditions, are sadly lacking.

Veterinary Research Findings and Future

It is evident from the above that veterinary research is perhaps the major area of importance for the future of wildlife utilization. At present there is no utilization and research directions have tended to concentrate on wildlife only in relation to their effects on domestic stock. Based on the information given in this paper as to the viability of wildlife for production and environmental preservation, and considering the Government's decision to open up this land-use system for the first time, there is a very great need for increased wildlife research.

It is with this in mind that I have followed with keen interest and personal association the work carried out in the Wildlife Diseases Section of the Veterinary Research Laboratories at Kabete. If wildlife is to assume its true position once again as the symbiotic partner in the grasslands of Africa, this research must be considered as vitally important to our future. The findings of this section led by Lars Karstad and Jan Grootenhuis are a pioneering start in a very wide, complex, and most important area.

It is my sincere hope that this project will continue. I am convinced it will have an increasingly important role to play in our future. The scope for research seems limitless at present. Basic parameters of normal or healthy animals are undocumented, let alone changes that come about due to diseases.

Conclusions

Wildlife research, particularly concerning health and transmissible diseases, assumes greater and greater importance as the prospect of genuine wildlife utilization systems develop. Questions such as stocking rates, species mix, including cohabitation with domestic stock, and general management practices are of increasing interest.

From the reasonable or logical point of view, if we are to look at conservation of our natural resources, particularly in dry lands, then we must find ways of keeping the true African animals. This cannot be done without their utilization, for if they cannot be used for emotional or political reasons or for lack of hard data, then they are a pest economically. They must be eliminated to make way for domestic stock that can be utilized, even if we are degrading our land and turning it to desert. Ways must, therefore, be found to utilize the African animals.

The advantages as outlined are many. We are dealing with a perfectly adapted group of animals and, provided we play the part of an enlightened predator, the system is self-perpetuating and non-destructive; thus, from the rancher's point of view, it would be a release from the struggle against nature. Rather, ranchers would be taking the sensible and easy road of cooperation.

From a national point of view, cooperation with nature is a question of survival for many countries. It is clear that the present paths are leading to catastrophy, loss of life, and loss of our basic resource, the land. The Sahel is the most glaring example, but the same is happening throughout the low-rainfall lands, Kenya included. If areas are developed for ranching, game will certainly begin to restore the natural balance of nature and increase the productivity of these areas. This system will restore the ability of the people to produce food and income.

It has become clear to me over 15 years of research and development that in land-use three considerations should be primary. First, the land-use system chosen should be as close as possible to the natural system. This will ensure its success and allow us to tap the resource and energy flow evolved over the millennia. Second, we should move always towards multiculture, for this is nature's way. Monocultures are proving to be less desirable and eventually uneconomic. Third, we must use systems that require no imported energy because these are expensive and, in the long term, impractical. Cattle ranching meets none of the above criteria. Game ranching, on the other hand, fits perfectly, and, I believe, it will prove to be the land-use system of the future.

What Ecologists Think Veterinarians Should Do[1]

Harvey Croze[2]

This paper will cover two subject areas: the relationship between disease and ecology; and the relationship between veterinary studies and ecological studies, all seen, of course, from an unbiased ecological perspective. I will also be presumptuous enough to point out what I feel are the broad gaps in wildlife veterinary research.

Ecologists are in general concerned with accounting for the distribution and abundance of plants and animals. This exercise often begins with the deceptively simple task of describing distribution and abundance, largely by drawing inferences from samples. Sampling strategies are designed to maximize the likelihood that the inferences are true and to minimize the cost of the sampling. With the basic inventory data collected and while monitoring changes and interdependencies, the ecologist then attempts to probe into causation.

The state of an ecological system at any particular time is the product of a number of processes, most fundamentally, birth and death. In ecological studies, the ecologist, even if primarily a botanist, must also attend to behaviour — from simple movements that can result in a particular instantaneous distribution or a rate of migration to complex social behaviour.

The ecologist likes to think of the task as a complicated one, especially when large numbers of species in whole ecosystems are considered. Figure 1 shows in an abbreviated way the relationship between the environment, basic ecological processes, and the end-products, abundance and distribution. The choice of "end-products" is biased toward those that may be most directly utilized by man — numbers of plants or animals in particular places. One could have picked as a goal for understanding something like social organization, population dynamics, or migratory behaviour; indeed, many respectable ecologists do just that. However, those interesting and important fields rarely lead directly to decisions that contribute to the well-being of people, unless the studies are extended to account for distribution and abundance.

In passing, it may be mentioned that the styles and rates of the processes shown or implied in Fig. 1 are modulated in part by species-specific characteristics of physiology and social organization. Physiological and behavioural adaptations are, like birth and death, determined by the environment, but they are moulded over an evolutionary time scale.

A decade ago, ecologists were faced with what may be called a crisis of scale. They began to realize that single species studies were necessary but by no means sufficient to justify expenditure of government resources and to provide decision-makers with information comprehensive enough to make the best decisions. In such a climate of necessity the ecological monitoring approach was born (e.g. Gwynne and Croze 1975, UNEP 1980). It combines intensive ground-based studies with extensive low-level aerial survey and satellite data collection techniques to understand as much as possible about areas as large as possible. Ground teams investigate the minutiae of primary productivity or animal population dynamics and provide "ground truth" for what the aerial teams observe.

The methodology is particularly well-suited to semi-arid ecosystems in which there are rapid and large-scale seasonal changes. On the one hand it covers in a cost-effective way the space in which the highly mobile populations of animals characteristic of such areas are likely to move about; on the other hand, the time-series nature and the diversity of the monitoring data allow correlations to be established between a number of ecosystem components that should lead to an understanding of causation. Such understanding is just one step away from rational management. To put it more simply, wildebeest, cattle, and pastoralists are dependent on water and grass, which in turn are dependent on weather, soils,

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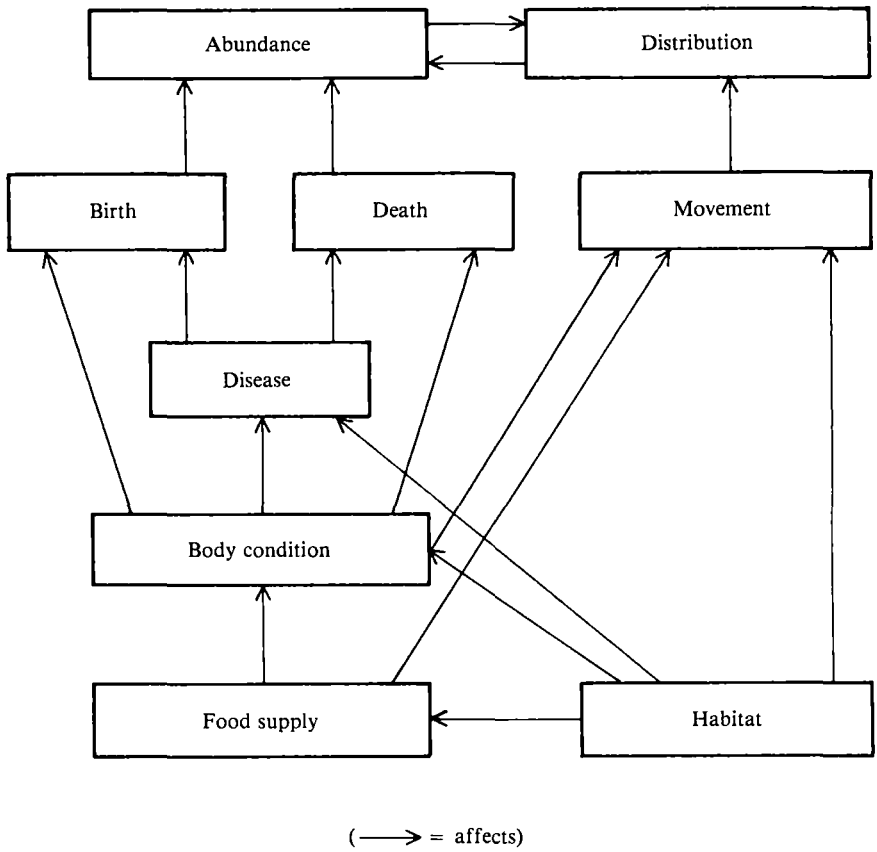

Fig. 1. Cause/effect diagram that shows why ecologists are (or should be) concerned with wildlife veterinary research.

and geomorphology. To know how the dependencies work and which components are most important in control of the system, one must measure them all simultaneously over time: one must monitor.

Where Disease Fits In

Disease, the domain of veterinary scientists, has a key position in Fig. 1. Disease obviously imposes a negative influence on the system: it slows down birth, increases the chance of death, decreases abundance, and restricts distribution. Such negative effects are not necessarily a bad thing, but rather part of the natural feedback mechanisms that regulate populations. Quantification of the dynamics of the diseases would allow ecologists to tighten up considerably their predictive population models, both for wild and domestic animals.

Disease in Fig. 1 is depicted as being dependent on food supply (because starved organisms are more likely to get sick than well-fed ones) and on habitat (because the distribution and abundance of a virus or of tsetse flies must be considered as characteristics of an organism's habitat, independent of food supply). Disease as conceived in the scheme is restricted to pathological processes and effects within the organism itself; pests and parasites are characteristics of the habitat, just as toxins would be products of the food supply. From this point of view, virologists and parasitologists could be considered as specialized ecologists.

Gaps in Veterinary Studies

It is difficult for ecologists to accept that disease may be as important as, say, food supply in determining the nature of ecosystems. It may well be so, but we cannot yet be sure for two reasons: (1) the nature of veterinary sampling; and (2) the lack of

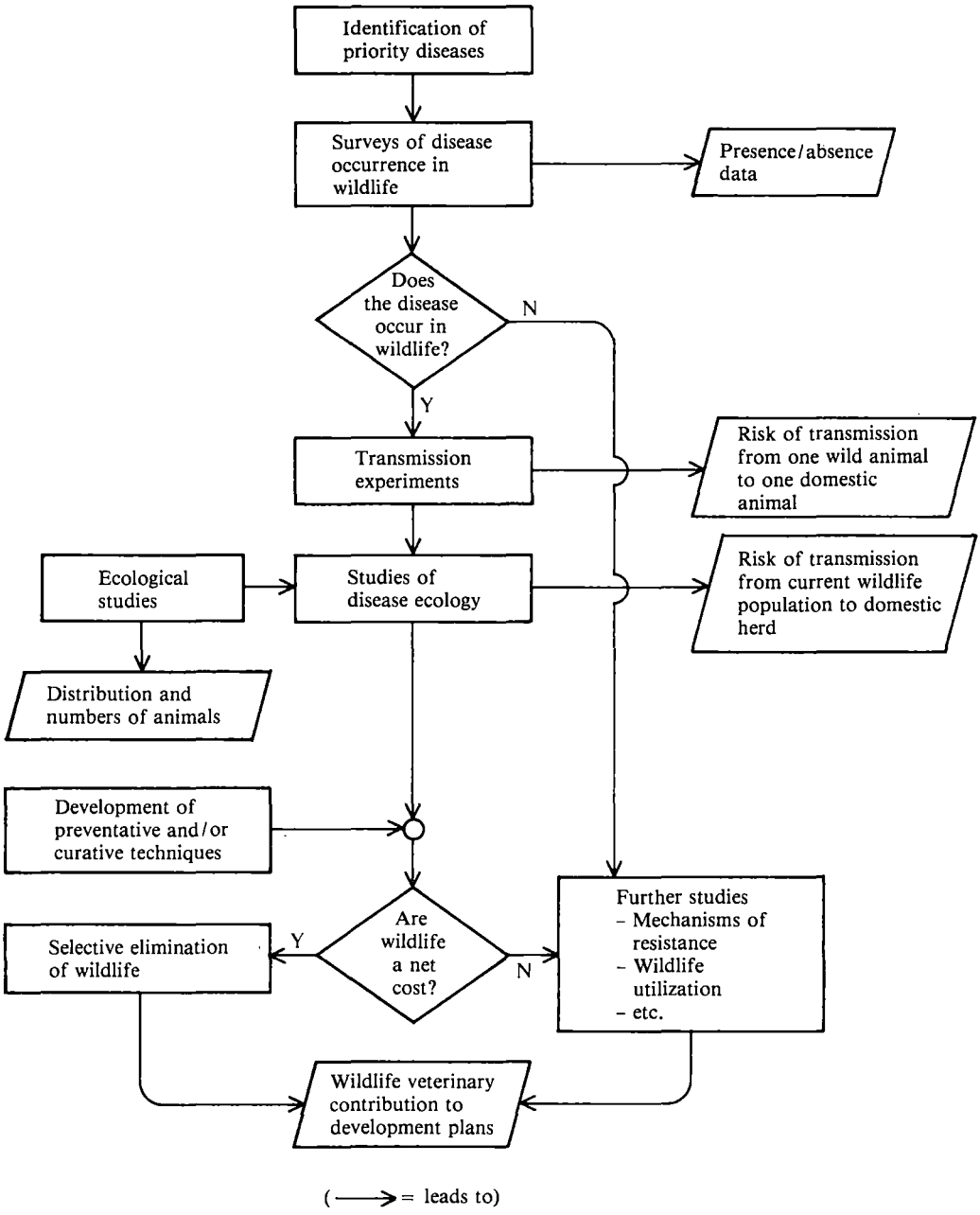

Fig. 2. A suggested framework for wildlife veterinary research.

integration of veterinary studies with ecological studies.

It seems to me that the theoretical base of veterinary sampling is weak or ill-defined. Perhaps my experience is limited, but I do not recall having ever seen a disease distribution map, laced perhaps with "isopaths" which would tell me the probability of a particular species contracting a particular disease at a certain place and time of year. Such a map would be as interesting and potentially useful to an ecologist as one of seasonal grass greenness. If it were overlaid on the seasonal distribution map of some

animal species, it might help to explain anomalies in the animal's distribution and abundance which were not accounted for by the distribution of food supply. Such an approach would help to make disease studies part of the whole, rather than an apparently disproportionately investigated part. It would indicate that the veterinary scientist was becoming an ecologist as well as a pathologist, a student of whole systems as well as one of individuals and organs.

I must admit that I am slightly foxed as to how such a metamorphosis would take place in practice, because the increase of investigational scale from the sick individual or its herd to whole populations in ecosystem contexts may be prohibitively expensive given currently available techniques. Nonetheless, there is a real need for rethinking veterinary sampling strategies and for developing rapid and extensive survey and diagnosis methods to produce information in study areas congruent with those now tackled by ecosystem ecologists. It is worth making the attempt because land use decisions and planning, particularly in semi-arid wildlife areas, are made at a scale that far outstrips the location of the individual or its herd.

One immediate approach would be to attach a veterinary subprogram to the ground work of ecological monitoring studies, and, who knows, to the aerial component as well. Already, the species, distribution, and density of carcasses observed from the air could provide stratification data for veterinary ground sampling. And, I wonder if a staring coat has special reflectance characteristics that could be detected on infra-red film, as is the case with diseased forest trees?

It should be stressed that I am suggesting augmentation to veterinary investigations, rather than replacement of classical studies that lead to such useful things as understanding of some effects of particular diseases, identification of pathways of transmission, and the development of control measures. The addition, involving a reexamination of the scale, scope, and methodology of sampling, would allow veterinary science to contribute to land use planning as surely as to prevention and cure of illness.

A Framework for Veterinary Research

Until wildlife comes into its own as a resource that can be utilized at the local as well as national level, it must be admitted that the raison d'être of wildlife veterinary scientists will be to show how their studies are useful to domestic animal husbandry. With this in mind, Fig. 2 represents a logical sequence of what one ecologist thinks veterinarians should do. It is a precedence diagram that leads from simple presence/absence statements of whether or not important diseases occur in wildlife (important in the sense of potential effect on livestock or man) to veterinary contributions to land use planning. Admittedly, the steps are grossly simplified, but they are an example of an orderly approach to the science.

Progress so far appears to be mainly confined to the first couple of boxes, that is, studies of occurrence and probability of transmission in quasi-laboratory conditions. Investigations of "disease ecology" that would lead to realistic risk assessments seem to be rather underdeveloped. Contrary to the logic of the scheme, research into prevention and cure technology seems in fact to have taken precedence over a basic understanding of the way disease systems work in the wild. I suppose this is because the demand to cure sick domestic animals and to keep healthy ones healthy is very pressing. Selective elimination of wildlife is probably necessary when they are proven reservoirs of harmful diseases and prevention and/or cure is costly. Otherwise, it is worth fighting the disease on a limited front, monitoring both its ecological and pathological effects, and using the wildlife in further studies, for example, to investigate alternative beneficial uses or to try and capitalize on their resistance mechanisms.

In conclusion, wildlife diseases are probably extremely important in the regulation of animal distribution and abundance. Wildlife veterinary research appears to have made good progress in what are viewed to be the preliminary stages of a comprehensive program that could contribute to rational land use planning and resource management. It is necessary to expand the scope of the research, to consider seriously the effectiveness of current sampling regimens, and to integrate veterinary studies with ecological research and monitoring.

Gwynne, M. D. and Croze, H. 1975. East African habitat monitoring practice: a review of methods and application. In Proceedings International Livestock Centre for Africa Seminar on Evaluation and Mapping of Tropical African Rangeland, Bamako, March 1975.

United Nations Environment Programme. 1980. Selected works on ecological monitoring. GEMS/PAC Information Series No. 1.

Discussion Conclusions

The Workshop provided an opportunity for those concerned with livestock ranching, animal health, wildlife conservation, ecology, and development planning to exchange views on the degree to which diseases of wildlife influenced development. The participants discussed not only the negative role of wildlife as carriers of diseases affecting domestic animals but also their positive contributions to the economy through their influence on tourist revenue, their effect on the ecology of rangeland, and their potential impact on meat supplies.

In Africa wildlife and domestic stock frequently graze the same lands. In looking at the interrelationships between these two groups of animals it was important to adopt an overall ecological approach that took into account both land use and resource conservation. In this context wildlife disease should not be viewed alone but as a component of a systems approach to ecology. Disease is a natural biological regulatory mechanism that helps preserve the stability of plant–animal complexes over large areas of Africa. When humans artificially distort this system, problems may become more prominent.

In economic terms domestic stock have historically been much more important to people than have wildlife. Thus, the economic rationale of controlling wildlife diseases has always been the impact of control on the health of domestic stock. In recent years, it has been recognized that wildlife can also have an economic role, contributing to tourist revenue and to meat supplies.

Wildlife have also been recognized as having a role in conserving grazing lands. Such conservation is of prime importance in Africa where the desert is advancing rapidly and taking over more than 60 000 km^2 of land every year. The 45% of the continent that is not already in desert is rapidly being destroyed largely as a result of human intervention. A declining area of usable land coupled with rapid population increase has resulted in the continent's requiring more than $550 million in food aid since 1973, and malnutrition is still rife.

The Workshop sought to examine the progress and results of wildlife disease research in recent years and to place these findings in an economic perspective. Its primary conclusion was that the role of wildlife as a carrier of disease of domestic stock had been overemphasized as a result of which much unnecessary slaughter of wildlife had taken place. It appeared that, to a large extent, domestic and wild stock can exist together harmoniously and that through appropriate strategies of land-use management the contributions to the economy can be synergistic rather than antagonistic.

It was considered that some of the research findings described at the Workshop had made a major contribution toward establishing the validity of these conclusions. Although further research is still necessary, many of the recent findings are conclusive. There is, however, a need to examine various aspects of wildlife policy in the

context of these disease relationships to maximize the returns from existing knowledge.

It was suggested that animal diseases in Africa can be divided into four categories: diseases that only affect domestic animals and not wildlife, e.g., contagious bovine pleuropneumonia and bovine babesiosis; diseases that exotic domestic animals had brought into Africa and now affect wildlife, e.g., rinderpest; diseases that were indigenous to African wildlife where they caused limited problems but that were of major importance in domestic animals, e.g., trypanosomiasis, African swine fever, African horse sickness, malignant catarrhal fever, and East Coast fever; and diseases that were confined to wildlife. Of these four groups it was clearly the third group that was of most interest in terms of the Workshop discussion.

Foot-and-mouth disease was one of the diseases that had received the most study, partly because of its high prevalence in domestic stock and partly because its existence acted as an important constraint to livestock exports. Research with wildlife had to date only isolated the virus from buffalo, although there was a suggestion that it is carried by the kudu in Botswana and that more extensive studies would reveal its presence in the warthog. However, even with buffalo it appeared that transmission to cattle was probably very rare. For example, in Zimbabwe Type SAT 3 was constantly found in buffalo but had not been identified in domestic cattle for 15 years.

In the past thousands of wildlife were slaughtered in rinderpest control programs. However, the disease is rarely seen in Africa today following an extensive Pan-African vaccination campaign. In the absence of disease in cattle it seems that it disappears from the wildlife population. Recently, however, antibody that neutralizes rinderpest virus has been found in several species of free-ranging wild ruminants. In the laboratories of the Kenya Agriculture Research Institute, a number of sera that had been collected in various parts of Kenya since 1975 and stored frozen at Kabete were tested. Seventeen of 147 sera neutralized rinderpest virus to a titre of 1/10 or higher in bovine kidney cell cultures. The positive sera came from buffalo, eland, impala, giraffe, waterbuck, warthog, and oryx, and their localities of origin were Kiboko, Nakuru, northern Turkana District, and Galana Ranch. To confirm that the neutralization was due to antibody, three of the sera were fractionated and the gamma globulin fractions retested. Neutralization occurred as with the whole serum. Sera from some jackals and hyenas also neutralized rinderpest virus, but it was thought that these species have cross-reacting antibody to canine distemper virus. It is not known whether this represents antibody to a strain of rinderpest of low virulence, or antibody to a rinderpest-related virus, such as that of peste-de-petits ruminants. When outbreaks of rinderpest do occur in cattle, it is probable that they arise from contact with infected cattle rather than from wild animals.

Malignant catarrhal fever has long been known to be transmitted by the wildebeest, although only recently has it been found that transmission is through contamination by ocular and nasal secretions of newborn wildebeest calves rather than from contact with the placenta or placental fluids. Antibodies to the causative virus can also be found in hartebeest, topi, and oryx, although no virus has been isolated from these species. At present, the only way of preventing the disease is by separation of cattle and wildebeest, at least for the 3 months following calving of the wildebeest.

There are a number of arboviruses affecting domestic livestock that have been isolated in East Africa, including the viruses of bluetongue and Nairobi sheep disease. These diseases often produce long interepizootics and do not seem to be of

great significance in wildlife, although they result in high mortality of domestic stock, particularly exotic animals.

Bovine petechial fever (BPF), a rickettsial infection, is probably also transmitted by an arthropod vector. The bushbuck acts as a passive carrier of BPF.

Trypanosomiasis and theileriosis are two diseases in which recent findings are very relevant to livestock development. Trypanosomes have been found in the blood of a number of wild species, although the infections caused by them are usually clinically inapparent. There appears to be a spectrum of tolerance to trypanosomes with the minimum being exhibited by exotic cattle, some tolerance occurring in the Zebu breeds, and considerable tolerance in the trypanotolerant Ndama breed and even more so in wildlife. It was suggested that there are certain nonspecific inhibitors in game sera (in the same way that human sera inhibit *T. brucei*) and that looking for such nonacquired serum factors in game and Ndama would be worthwhile. There also appears to be a promising potential for the ranching of species such as the oryx in areas where trypanosomiasis is endemic.

With regard to theileriosis it appears that parasites of this genus can be found in the blood of most wild ungulates. The only *Theileria* species that appears to cause problems in domestic cattle is the very virulent *T. lawrencei* from buffalo. There is some suggestion that *T. parva* evolved from *T. lawrencei*, but the precise relationship between the two species still needs to be determined. It was also pointed out that "transformation" in the case of *T. lawrencei* Serengeti seems to be a stable, enduring change.

Intestinal helminths of wildlife appear to be only rarely important parasites of domestic ruminants. However, there is a suggestion that their presence interferes with the success of the modern approach to immunization of sheep against worms such as *Haemonchus*. It was pointed out that in the event of attempts to vaccinate sheep against *H. contortus* the contamination of pastures by gazelles would become a matter for concern, because early exposure of lambs to *H. contortus* results in a state of immunologic unresponsiveness when sheep are inoculated later with a vaccine consisting of irradiated larvae. To produce a good vaccination-induced immune response, lambs must not be exposed to *H. contortus* early in life.

In East Africa cysticercosis and echinococcosis present problems of public health importance. The former condition appears to be common in game meat and could represent a constraint to game ranching. Echinococcosis is extremely widespread in certain tribal grazing areas and appears to be primarily attributable to contamination by the large populations of dogs. In Turkana wild animals do not appear to be important as carriers of the disease and its incidence could be significantly reduced if the stray dog population were reduced and the valued dogs were regularly treated with anthelmintics.

The finding that harmonious coexistence of domestic and wild stock is a feasible proposition is important from the standpoint of land use. Certain high-risk situations have been clearly identified such as the risk of MCF through contact with wildebeest and *T. lawrencei* infection from buffalo. However, in general, the disease risks from wildlife seem to be limited and the prospects for a more effective and economic utilization of wildlife appear to be promising. For instance, one species of game animal that is being tried as a meat production animal is the oryx; in a study from July through September 1979, blood samples were taken from 50 tame fringe-eared oryx (*Oryx biesa callotis*) along with 50 yearling Boran steers, and 50 young goat

wethers located in the same general area. The complement fixation test, subinoculations of blood into mice, and microscopic examinations of fresh wet mounts of the "buffy coat" leukocyte fraction, along with thick and thin Giemsa-stained smears, were used to survey for antibody and infection in the three groups of animals. Both the steers (30%) and the goats (12%) showed evidence of one or more species of trypanosomes, namely *Trypanosoma vivax*, *T. congolense*, and *T. brucei*; however, there was *no* evidence of trypanosomiasis in any of the 50 oryx. A follow-up study indicated that about 10 times as many tsetse flies were attracted to calves as were attracted to the oryx.

For the prospects of game utilization to be realized some important changes in traditional attitudes toward wildlife will need to be implemented. Paramount among these changes is the need to regard wildlife as an economic resource and to recognize that only to a very limited extent do wildlife diseases hinder the development of the domestic livestock industry.

Such a change in attitude is an essential prerequisite for initiating appropriate legislation to encourage the development of better wildlife utilization through the introduction of appropriate management systems such as game ranching. For example, in Kenya, it is a paradox that it is currently illegal to harvest meat from indigenous African species raised on African lands.

The scope for wildlife ranching is immense because this appears to be the only alternative form of land use in arid range areas that are degraded by cattle. It has been shown that, in circumstances in which cattle destroy the vegetation, gazelle are a feasible alternative. In certain circumstances they may produce more meat per land unit, and some species, such as oryx, can live where the available water supply is too low for cattle to survive.

Research on wildlife diseases has shown that much of the opposition to wildlife is emotional. There are, nevertheless, obstacles to the farming of wildlife such as the definition of ownership and the costs of preventing poaching, and steps will need to be taken to overcome these problems before game ranching is likely to become widespread. Research will also be needed to define the most appropriate economic balance between different systems of wildlife utilization such as ranching and hunting.

It is recognized that wildlife disease research can be expensive and time-consuming and considerable thought is necessary to define the priorities for such research in terms of both diseases and species. It appears to be particularly important to expand the scope of this research to take into account the current weakness in sampling regimens that can distort the value of the epidemiologic findings. Thus, it would be desirable for those responsible for research on wildlife disease to work more closely with those responsible for ecological research and monitoring. The work entails combining intensive ground-based studies with extensive low-level aerial survey and satellite data-collection techniques to produce as much information as possible about animal–plant relationships in as large an area as possible. The wildlife disease specialist has an important role to play in this undertaking.

To fulfill this role there is a need for wildlife disease research to be ongoing. The experience of the last 5 years in Kenya suggests that it can be highly successful when conducted at a low level of staffing, providing the unit liaises effectively with others working in the same and allied disciplines. The level of participation in the Workshop suggested that the existing team had been very successful in this respect.

For wildlife disease work to realize its full economic potential in East Africa, it will be necessary for policy and legislative changes to take place. The Workshop participants considered that the climate for such changes was good among those most intimately concerned with the subject. There is, however, a great deal of emotional pressure from outside Africa from those who do not appear to comprehend that rangeland conservation and wildlife conservation go hand in hand and that a rational land conservation strategy includes the management of wildlife numbers. There is a need for those with an understanding of the animal–human–land relationships to stand up to these pressures if a rational conservation strategy is to evolve.

With many of the diseases mentioned above, sufficient knowledge already exists for effective control programs to be established that would facilitate the harmonious coexistence of domestic and wild stock. Much relevant information has been provided during the last 10 years through the collaborative activities of the Wildlife Diseases Section at the Veterinary Research Laboratories in Kabete. This unit, with a very small professional staff, has successfully functioned as a coordinating centre with a large number of other organizations and laboratories working on specific diseases. In this way a limited amount of wildlife material has been used for a large number of disease investigations. It was a significant feature of the Workshop that, although very few papers were presented by staff of the Wildlife Diseases Section, virtually every author acknowledged the unit's collaboration. It appears that there is considerable merit in a small wildlife disease research unit functioning in this way and collaborating with units specializing in specific diseases. There was a consensus at the Workshop that this approach was highly cost-effective.

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